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AN EXPERIMENTAL AND CLINICAL STUDY

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THE UNDESCENDED TESTIS

AN EXPERIMENTAL AND CLINICAL STUDY *

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MINNEAPOLIS

It is now generally believed that the undescended testis is usually incapable of a continued production of spermatozoa. Hunter,** [123] who first described the normal descent of the testis, stated that failure of the testis to reach the scrotum is due to its initial imperfection. Most European investigators hold to this opinion. But operations are continually being performed on the undescended testis in the hope that with transference to the scrotum that spermotogenesis will occur. If the development of the testis is not altered by transition to the scrotum, only the belief that the danger of malignancy is lessened or that the patient is more pleased with his testis in its usual habitat would justify operative interference in the absence of pain or other complicating factor. A decision as to whether the testis is ab ovo deficient or whether its abnormal situation is responsible for its lack of development is therefore of paramount importance in the rational treatment of the undescended testis.

Orchidopexy is an old procedure and it would seem that the examination alone of those cases in which the operation has been done for failure of both testes to descend would determine whether or not an undescended testis would develop normally if placed in the scrotum. Records of results are numerous, as concerns the position of the transplanted organ, and many observers [16, 43] record an increase in size after scrotal fixation. But reports concerning examination of the semen when the operation has been done for double incomplete descent are unfortunately few, so few in fact that it could with just cause be claimed that these are merely examples of those rare instances in which a temporary spermatogenesis occurs in cryptorchids.

A sufficient number of such cases have now been recorded that one cannot deny that spermatogenesis [250] may occur in cryptorchids. Marechal [174] in 1887 was able to compile fifty cases in which histologic examination of the removed organ or semen examinations in double incomplete

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** Dr. R. E. Scammon informs me that Zerkis in 1495 had previously described the descent of the testis, but the first adequate description was given by John Hunter.

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descent disclosed the presence of sperm. Taylor [266] in discussing the medicolegal aspect concerning the fertility of cryptorchids enumerates a number of cases in which the procreative power of such persons has been unquestionably established. A few writers would have us believe [42], [281] that it is the rule for testes retained within the abdomen to be quite normal. Uffreduzzi [277], [278] says that 10 per cent of cryptorchid testes exhibit spermatozoa. Rawlings [217] states that in fifty undescended testes removed at operation twenty-seven were examined microscopically and of these ten were fairly normal and showed a definite spermatogenesis. Odiorne [201] and Simmons examined seventy-seven postpuberty undescended testes and all of these showed degenerative changes. They report one instance of fertility in a man of 20 when neither testis was in the scrotum. Bland-Sutton was only able to find spermatozoa once in the histologic examination of twenty-five incompletely descended testes. Lanz [158] saw spermatozoa once in eleven testes that failed to reach the scrotum. Griffiths [112] doubts the authenticity of every case in which spermatogenesis is said to have occurred in bilateral incomplete descent, and doesn't believe that the retained testis can produce spermatozoa. In practically every instance in which speramatozoa have been demonstrated in the examination of cryptorchids the subject has been young.[286] So constant has this finding been that one theory as to the nature and behavior of the undescended testis depends for its explanation on this factor.

Monod and Arthaud,[187] on the basis of finding a fairly normal histology in the undescended testis of a man of 20 and atrophy in two men over 40, promulgated the idea that incomplete descent is compatible with fecundity in the young but that with increase in age an atrophy slowly and progressively occurs.

Two other opinions as to the condition of the incompletely descended testis have been given credence by those who have investigated this problem. Curling [64] held that undescended testes developed normally to puberty, then failed to undergo the changes incident to this age and remained in the prepuberty stage. Histologic study, however, demonstrates that a real degenerative process is observed in the ectopic testis with the supervention of puberty.

Hunter [130] believed that the aberrant position and absent spermatogenic function were both due to congenital imperfections and that "nothing can be done by art to give the testicles the stimulus of perfection which is necessary to make them assume the disposition requisite for their descent." Hunter's opinion today claims as adherents numerous supporters and investigators. Bland-Sutton [22] states, "A testis is retained because it is imperfect. The migratory impulse in a healthy, normal testis is irresistible"; and again, "The imperfections of an undescended testis are the cause, not the consequences of its failure

to reach its goal in the scrotum. . . . Surgical efforts to preserve a retained or a partially descended testis may be described as supererogation." At a recent meeting of the Royal Society of Medicine in London, Bland-Sutton [24] reiterated his previous stand and said that "the notion that the functional imperfection of a retained testis is due to its nondescent is false." Eccles [76] at the same time said that it was wrong to speak of atrophy and that an undescended testis is underdeveloped Finotti [94] also shares this opinion. Uffreduzzi [277] describes nondescent as a local expression of infantilism. Recent investigations on the continent seem to bear out Hunter's theory. Staemmler [254] believes that a hypoplasia of the spermatic arteries is the cause of lack of development and also prevents descent. Schinz and Slotopolsky [234] state that cryptorchidism is essentially a hypoplastic process and that this hypoplasia is only a part of a general developmental disturbance "vitium primae formationis."

It has, however, long been felt by many that given the advantage of the natural environment that the scrotum affords an incompletely descended testis would develop normally and that the success or failure of the scrotal fixation would be a dominant factor in determining the future possibilities of the imperfectly descended organ. The almost universal practice of orchidopexy therefore signifies that many still cling to the belief that failure to reach the scrotum accounts for the aspermatic condition of the undescended testis.

Is has been my object in this study to investigate the merits of these two controversial opinions. If the incompletely descended testis owes its imperfect development to its failure to descend, a successful orchidopexy would rectify the condition and stamp the operation as being of material and functional benefit. Failure of the germinal epithelium to develop would indicate that the object of the procedure is only cosmetic. These two diametrically opposed ideas cannot be compromised. Does transition to the scrotum enable an imperfectly descended testis to develop normally or is it *ab initio* imperfect and incapable of mature growth?

MODE OF INVESTIGATION

In the study of this problem several testes that failed to reach the scrotum and were removed in the course of operations for hernia in adults have been examined. Normally descended testes removed in the radical cure of large hernias and hydroceles have also been submitted to examination. Testes of young pups and adult dogs have been placed in the peritoneal cavity and the inguinal region and the effect on the organ noted. After having been allowed to remain in the peritoneal cavity or inguinal region for a given period, a number of

testes have been redeposited in the scrotum. The influence of severance of the blood vessels in the spermatic cord and ligation of the vas deferens has also been noted. Special study has been given the anatomy of the prepuberty testis.

DESCENT OF THE TESTIS

The abdominal position of the fetal testis was first mentioned by Haller.[130] Frankl [98] states, however, that this was also known to Galen. Hunter,[130] who first described the normal descent of the testis, was at a loss for a satisfactory explanation. Many of the early investigators attributed it to the action of the cremaster.[270] Edwards [77] says that the gubernaculum shortens with descent and turns about much as can the finger of a glove. Hart [24] speaks of the gubernaculum as a rudder. Keith * states that the gubernaculum in drawing the peritoneum down drags the testis with it like a log on a sledge. That the gubernaculum has little or no active part in the descent of the testis becomes evident in the cases in which the testis is in the scrotum, but there is an absence of fixation [279] to the scrotum by the gubernaculum.

The testis develops on the inner side of the wolffian ridge in close relation with the kidney and remains intra-abdominal in position [29] throughout the greater portion of fetal life. Scammon [232] divides the transition of the testes to their scrotal position into three stages. In the sixth week of fetal life, the genital ridge extends from the sixth to the twelfth dorsal segments. During the third month, because of an atrophy of the cranial segments, the definitive testis lies in the iliac fossa. As a consequence of unequal rate of growth of structures below and above the inguinal ligament, the testis comes to lie at the future internal abdominal ring. These two phases of descent are entirely passive. Preceded by the vaginal process of peritoneum, the testis passes obliquely through the abdominal wall, reaching the external abdominal ring during the seventh or eighth month. In over 90 per cent of new-born children the testis has arrived at the bottom of the scrotum. The scrotum, a derivative of the abdominal wall, is preformed to receive the testis. At birth the upper portion of the vaginal process is obliterated,† but the lower end persists throughout life as the tunica vaginalis propria. Failure of this peritoneal tunic to obliterate in the upper portion constitutes a potential inguinal hernia. The passage of the testis through the abdominal wall is an active migation, but Scammon [232] states that there is no known cause adequate to account for this stage of descent.

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* Human Embryology and Morphology, New York, 1921, p. 399.

† For an excellent discussion of the obliteration of the vaginal process reference should be made to Engel,[81] Frankl,[98, 99] Lockwood,[169] Sachs [228] and Zuckerkandl.[302]

The testis may be arrested in its descent at any point, most frequently in the inguinal region and almost as often at the external abdominal ring. It may remain in the abdominal cavity in the iliac region or descend into the perineum. Femoral ectopy of the testis has been described but Eccles [72, 73] considers its existence doubtful. He states that in almost every instance the testis passes through the canal and passes into the upper portion of the thigh through the external abdominal ring. In transverse ectopy of the testis both organs occupy one side of the scrotum. This abnormality may be due to faulty development or faulty descent. In event the former condition obtains both testes develop on the same side; in the latter they descend through the same inguinal canal but develop from genital buds on opposite sides. This abnormality of descent was first described by Lenhossek.[162] Kimura [143] compiled ten such cases from the literature and reported another in 1918. Pubopenile ectopy of the testis [134] is also very rare. Two cases [95, 197] have recently been reported. Perineal ectopy has been ascribed to a vicious insertion of the gubernaculum, and to being pushed into an abnormal position by a hernia. Weinberger [289] in 1899 was able to find seventy-three cases of this type. He did not believe that the action of the gubernaculum accounted for the descent of the testis into the perineum. In 1906 Klein [144] reported eighty-one cases of perineal testes. According to Sonneland,[251] the superficial inguinal type of interstitial ectopy, the perineal and the pubopenile may all be accounted for as types of incomplete descent in which the testis has passed out through the external abdominal ring but failed to reach the scrotum. In interstitial hernia the testis may be carried up over the abdominal wall above or beneath the external oblique aponeurosis or less commonly beneath the internal oblique. Krönlein [149] early pointed out the frequent association of incomplete descent of the testis with this type of hernia. Eccles [74] states that in 73.4 per cent of cases arrest of descent is associated with inguinal hernia.

Ectopy or an abnormal situation outside of the usual path of descent is considerably less frequent than incomplete descent. Monod and Terrillon [188] ascribe to Kocher this differentiation between ectopy and incomplete descent.

FREQUENCY OF UNDESCENDED TESTES

Statistics of the Austrian army [301] show that for every thousand men drafted for military service, 2.2 have an undescended testis. Rennes [220] observed six cases amongst 3,600 recruits. Marshall [177] found eleven in 10,800. The war department medical records [287] for the World War show that there are 3.1 cases of incom-

plete descent * for every thousand cases examined. Double incomplete descent [188] is incomparably more rare than single and right sided arrest of descent occurs more commonly than it does on the left. This is in agreement with the fact that the left testis usually descends earlier than the right.

Only rarely does the testis reach the scrotum by its own initiative if it has failed to do so at the end the first year. Neugebauer reports thirty-eight cases of late descent.[128] With the increasing obliquity [40] of the inguinal canal due to the growth of the pelvis, the possibility that the testis may migrate spontaneously to the scrotum decreases greatly. Occasionally a testis that has been in the scrotum will return to the inguinal region. This is known as "ectopie en retour." [128] Stefko [257] has recently described the finding of a high percentage of inguinal testes in starving children (27 per cent). He attributes this finding to too short a cremaster occasioned by starvation in which an unequal growth of the muscles of the abdominal wall occurs.

CAUSE OF FAILURE OF THE TESTIS TO DESCEND

Undoubtedly a number of factors may operate to bring about incomplete descent of the testis. Eccles [75] classifies the conditions that may be causative as follows:

1. Conditions associated with the mesorchium.
2. Those associated with the testis and its component parts.
3. Conditions associated with the gubernaculum.
4. Conditions associated with the cremaster.
5. Those associated with the route along which the testis must pass.

In by far the majority of cases of inguinal retention, however, separation of the spermatic cord from the processus tunica vaginalis testis suffices to mobilize the gland so that it may be placed in the bottom of the scrotum.[25, 45, 171, 212] Bevan [16] states that separation of the peritoneum from the vas deferens and spermatic vessels, if carried from 2 to 3 inches (5 to 7.6 cm.) within the abdominal cavity and combined with removal of all the coverings of the cord, so that the testis hangs suspended by the vas and vessels alone, will so mobilize the testis that in many instances it may be placed on the thigh several inches below the inguinal ligament. Bevan [19] says that in only 10 per cent of cases has it been necessary to section the vessels in the pampiniform plexus in order to bring the testis into the scrotum. Büdinger [38, 39] states that the vessels are always short and cites the relation that obtains in

* This figure includes anorchism and monorchism. Absence of the testis is an exceedingly rare anomaly and it is scarcely possible during life to obtain reliable proof concerning its existence. Jacobsen [134] says that Gruber in 1868 could compile only twenty-three cases of unilateral anorchism and seven of the bilateral variety, all having been verified by necropsy.

ectopic kidneys. The vas deferens is always long enough he finds, but the vessels are no longer than they need to be for the position of the testis. The most common cause of retention of the testis he ascribes to peritoneal adhesions [38] occasioned by fetal peritonitis or inflammation in early postembryonic life. Bramann [30] and Robitansky have each observed a case in which shortness of the vas prevented the testis from reaching the scrotum. According to a few investigators, [106, 277] heredity plays a dominant rôle in bringing about nondescent of the testis.

THE ANATOMY OF THE NORMALLY DESCENDED TESTIS AND OF THE UNDESCENDED TESTIS

The testes are bluish white ovoid bodies suspended in the scrotum (about 25 Gm. each in weight). The tunica vaginalis testis ensheaths the whole testis anteriorly: on its posterior aspect where this tunic is deficient the testis is capped by the epididymis. Here also the vessels leave the testis. Beneath the visceral lamina of the vaginal tunic investing and fused with the testis is the tough tunica albuginea. From the deep surface of this covering a number of slender fibrous bands dip into the gland. These septula testes imperfectly divide the parenchyma [71] into a number (from 100 to 200) of wedge shaped lobules whose apexes converge toward the mediastinum testis at the posterior border of the gland. The parenchyma of the organ is composed of large numbers of manifoldly twisted tortuous seminal canals which may anastomose [31] with one another. These, the seminiferous tubules (about 600) completely fill up the spaces between the septums and are loosely held togther by connective tissue. At the apexes of the lobules they unite to form the straight tubules, short narrow seminal canals which open into a complicated network of fine channels known as the rete testis in the mediastinum. Out of this network from about fifteen to twenty minute tubules,[71] the efferent ductules of the testis, pierce the tunica albuginea at the upper end of the posterior margin and enter the caput epididymidis. After an unusually tortuous course, these ducts end at the cauda epididymidis by becoming the vas deferens. The latter, the excretory duct of the testis, begins as the continuation of the ductus epididymidis and ends by uniting with the excretory duct of the seminal vesicle to form the ejaculatory duct emptying into the urethra in its posterior portion.

The minute anatomy of the testis shows it to be a tubular gland with a loose interstitium separating the tubules. Each seminiferous tubule in the adult testis contains several layers of epithelial cells. The Sertoli or nourishing cells lie directly on the basement membrane of the tubule. They have no part in the spermatogenic cycle. They are elongated irregularly pyramidal cells [71] with large oval nuclei rich in chromatin. The cytoplasm of these cells has a high fat content.[118, 119] Toward the periphery

of the tubule and scattered between the Sertoli cells are the spermatogonia, the progenitors of the adult male sex cell. In the prepuberty testis, the Sertoli cells and the spermatogonia are the only cells lining the seminiferous tubules. In the mature testis, the spermatogonia are small round elements with nuclei rich in chromatin. By growth the spermatogone becomes a spermatocyte of the first order. These divide and form two secondary spermatocytes whose division gives rise to the spermatids from which the spermatozoa takes origin.

In the intertubular spaces there are groups of cordlike masses, the interstitial cells.* These are rounded polygonal elements with small eccentric nuclei. Scattered in the finely granular cytoplasm may be found pigment particles or crystalloid bodies [211] in the form of minute rods or needles.

The main artery of the testis is the internal spermatic which takes origin from the abdominal aorta. In addition it is also supplied by the deferential and external spermatic arteries which establish anastomoses with the internal spermatic. The testicular branches enter the mediastinum and break up into superficial and deep twigs that follow the tunica albuginea and the septums, respectively. The latter follow the tracts of intertubular connective tissue and ultimately form rich capillary networks enclosing the seminiferous tubules outside the basement membrane. The veins arise in these networks and emerge at the mediastinum and together with the veins from the caput epididymidis ascend in the spermatic cord in the anterior part of the pampiniform plexus. The veins from the body and tail of the epididymis unite into a smaller group that ascend in the posterior part of the plexus.

The lymphatics of the testis follow the course of the veins and drain into the lumbar lymph nodes. The nerves of the testis are chiefly sympathetic fibers that follow the course of the internal spermatic artery.

The histology of the immature testis and the blood supply of the organ will be dealt with more fully later.

The retained testis of the adult when compared with its normally descended fellow, though preserving the contour of the normal organ, is of much smaller proportions. But apart from this disproportion in size the gross anatomy of the normal organ is preserved.[91] Occasionally the body and tail of the epididymis remain separated from the testis by the mesorchium.[39, 140]

When examined microscopically, however, a remarkable difference in structure is at once apparent. The singular disappearance of the germinal epithelium from the seminiferous tubules is most striking.

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* Odiorne and Simmons [219] and Coley [49] state that there are no interstitial cells in the normal adult testis. Winiwarter,[293] however, finds them present and Benoit [13, 14, 15] states that failure to observe them is due to lack of proper tissue fixation.

Frequently only the bare outline of the tubule persists as a hyaline ring. Many have a single layered epithelium constituted only by the Sertoli cells. No spermatogonia or other sex cells are usually present. In the retained testes of individuals just past puberty, however, spermatogonia and spermatocytes are not infrequently found. In seventeen undescended testes that I examined microscopically, spermatozoa were never observed. But in several fairly young subjects spermatogonia were often present. The number of prepuberty undescended testes that have been examined are, as might be supposed, very rare. In the instances in which this has been done, however, the degenerative changes that occur in the post-puberty testes are absent and the histology of the gland is much the same as a normally descended organ of a similar age.[20, 86, 89] This matter will be discussed at more length in the consideration of the anatomy of the immature testis. The interstitial cells in the atrophic adult incompletely descended testis appear increased in number. It has been felt that this apparent increase is real, but recent quantitative measurements by Bascom [9] would indicate that no real increase occurs.

COMPLICATIONS TO WHICH THE UNDESCENDED TESTIS IS LIABLE

Pain.—The incompletely descended testis is subject to the same diseases that affect the normally placed organ. Failure of the testis to descend completely may give rise to no subjective symptoms. Inguinal retention of the testis may be associated with pain. The abnormal position in such an event renders it especially liable to traumatism and recurrent attacks of pain due to this cause are not infrequent.[286]

Hernia.—It is usually an associated hernia that urges the patient with incomplete descent to seek medical aid.[53] An inguinal hernia may occur if the testis is retained within the abdomen because the processus vaginalis testis [29, 128] precedes the testis in its normal descent into the scrotum. Coley [51] states that in 80,736 cases treated for hernia 1,357 were complicated by undescended testes. Eccles [73] reports 854 cases in which arrest of descent was present in 48,000 cases of inguinal hernia. Uffreduzzi [277, 278] states that in 90 per cent of cases of incomplete descent there is an associated hernia. Schönholzer [238] finds a hernial sac present in 93 per cent; Rawlings in 75 per cent; [217] Odiorne and Simonds [201] in only 57 per cent; Rovsing [227] in 100 per cent of cases of failure of the testis to complete descent. In probably every case there is a potential hernia due to a persistent tunica vaginalis testis.

Torsion.—Nicoladani * called attention to the fact that the undescended testis is more liable to rotation of its pedicle with torsion and subsequent strangulation than the normally descended organ. In 1901

* Nicoladani: Arch. f. klin. chir. **31**:178, 1885.

Scudder [242] compiled thirty-two cases of torsion of the pedicle of the testis. Forty-seven per cent of these occurred in undescended testes.

Malignancy.—Nearly all who have written on the subject of malignancy in the testis are agreed that the incompletely descended organ is more likely to become malignant than the scrotal testis. Malignancy of the testis is a rare disease.[21, 237] In 1922 Tanner [265] stated that only about 600 cases had been reported in the literature. Of 452 cases compiled by Cunningham,[63] 412 had occurred in the normally descended testis. Malignancy in this series therefore was 10.3 times more frequent in the scrotal than in the undescended testis. When, however, allowance is made for the fact that only one testis in 500 is undescended, it is apparent that in Cunningham's [63] series, at least, the undescended testis is fifty times more likely to malignant degeneration than is the normally descended testis.

Tanner [265] believes that the testis retained within the abdomen is less likely to become malignant than is the normally descended testis. Bulkley [41] states that about one testis in seventy-five retained within the abdomen will become malignant, and that one out of every four malignancies in undescended testes is found in the abdomen. Several believe that the testis within the abdominal cavity enjoys an immunity from malignancy and that when an inguinal testis cannot be placed in the scrotum that it can with safety be placed in the abdominal cavity behind the peritoneum.[128, 238] A few writers state that this is the operation of choice in the treatment of undescended testes,[159, 281] and that an imperfectly descended organ is as likely to function there as in the scrotum. Uffreduzzi [278] and Keyes and MacKenzie [142] state that the abdominal testis is more likely to become malignant than the testis retained in the inguinal region. Malignancy has never been observed in a perineal testis.[144]

No greater diversity of opinion probably exists concerning anything in medicine than the question of malignancy in the undescended testis.

The opinion prevalent today that the abdominal testis is less prone to become malignant than an inguinal testis or one normally situated I believe to be but a heritage from earlier literature.[84, 137, 256] In 1893 Farwick [84] could find only three cases of malignancy in the testes retained within the abdomen and seventy-seven in inguinally retained testes. In 1901 Kaeppelin [137] could find only six malignant abdominal testes. Bulkley [41] in 1913 reported fifty-nine cases of malignancy in the abdominally retained testes. Cunningham [63] in 1921 stated that three had been reported since the appearance of Bulkley's paper. I have observed two cases of malignancy in the abdominal testis. Malignancy of the abdominally retained testis in the male hermaphrodite is not infrequent. Zacharias [300] mentions thirteen cases, two of which were bilateral.

Bland-Sutton [24] says that thirty-five have been reported. Romiti [225] reports the case of a man at thirty-seven who had a malignant abdominal testis. Eleven years previously during the course of a hernia operation the testis which had previously been scrotal in position was placed in the retroperitoneal region in order to better treat the hernia.

When it is recalled that malignancy is a rare disease in the testis, that an undescended testis is also infrequent, and that abdominal retention of the testis occurs with considerable less frequency than does inguinal retention, it must be conceded that an abdominal situation of the testis is not a protection against malignancy.

It has long been felt that the exposed position [70, 245] of the inguinal testis to trauma renders it susceptible to malignant change. The relative frequency of torsion in the inguinal testis has been pointed out. Kahlden [138] believes that the contraction of the abdominal muscles over the testis in the inguinal region, as occurs with coughing, lifting, etc., is responsible for the frequency with which malignancy is observed in the inguinal testis. Landau states that the contraction of the abdominal muscles over an inguinal testis could exert but little pressure on the gland.[277]

Coley [52] believes that trauma is a factor in one third of the cases of malignancy of the testis. Chevassu [47] states that traumatism favors the growth of a tumor but believes that trauma as a causative factor in testicular malignancies has been over rated.

In the undescended testis, the anatomic peculiarities * observed are the disappearance of the germinal epithelium and a hyperplasia of the interstitial cells. It has been suggested that the relative frequency of malignancy in undescended testes takes origin in these cells,[62, 29] but sarcoma of the interstitial cells [83] is a very rare tumor. A benign tumor † arising in the interstitial cells is not uncommon.

Wilms [292] stated that the complex malignant tumors of the testis contained all three germinal layers and were best considered as teratomas. Ewing [82] from a careful study of nineteen cases and an examination of the literature on the subject states that "the great majority or possibly all of these tumors are of teratomatous origin." Ewing [82] had several thousand serial sections made of embryonic testes through the corpus Highmori and rete testis. Several islands of chromaffin tissue and two misplaced groups of squamous epithelium were found. Ewing concludes "that teratoma testis arises from sex cells in the neighborhood of the rete, whose normal development into spermatogonia has been suppressed but whose potencies remain intact or ready to express themselves in the various forms of simple or complex teratomata."

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* It has erroneously been maintained that an ectopic perineal testis [28, 203] is histologically normal.

† It is said to occur frequently in old dogs.[108]

The relative frequency of malignancy in the undescended as compared with the normally descended organ is best accounted for in Ewing's explanation of the mode of origin of malignant testicular tumors.

Prophylactic removal of an inguinal testis because of the danger of malignancy is scarcely warranted. Such a procedure has not been applied to other organs in which malignancy is manifoldly more common.

Several instances have been reported in which tumors have occurred in the normally descended testis while the same patient carried one in which only partial descent had occurred. Howard [129] records the instance of a man of 37 the subject of a carcinoma of the testis. At 7 his testis was in the scrotum; as a result of trauma dislodgement occurred into the inguinal region. The converse has also been noted. Cunningham [63] reports the case of a man of 30 the subject of a teratomatous testis. At 25 this testis had been in the inguinal region and at that time was placed in the scrotum. Coley [51] reports an instance in which malignancy of the testis followed closely on replacing the testis in the scrotum. Tyrell [276] mentions the case of a man whose left testis descended into the scrotum spontaneously at 20. Twenty-five years later, at 45 years, a sarcoma developed in this testis. Marlier [175] mentions such an instance in a man whose left testis first descended into the scrotum at 16; at 38 the testis was the seat of a carcinoma. Romiti [225] relates the history of a man of 37 who developed a malignancy in a testis once resident in the scrotum that eleven years previously was placed in the abdomen in order to cure a large inguinal hernia.

Orchidopexy therefore does not diminish the possibility of a malignancy developing in the testis with reposition of the organ into the scrotum.

EXPERIMENTAL INVESTIGATION

All experiments were done on dogs (a few early experiments not recorded were done on guinea-pigs and rabbits) under usual aseptic technic. Ether was used to obtain anesthesia. Fine linen sutures were used throughout. The experiments will be tabulated according to type of procedure instead of in chronological sequence. The examination of the undescended testes and the testes removed in the course of operations for the cure of hernia and hydrocele, and those obtained from persons dying of acute febrile and chronic debilitating diseases will be recorded following tabulation of the procedures on dogs. Formaldehyde fixation (10 per cent) of tissue and hematoxylin and eosin stains were used. All tissue was embedded in paraffin and the sections were cut from 8 to 10 microns in thickness.

TRANSPLANTATION OF TESTES OF MATURE AND YOUNG DOGS INTO THE ABDOMINAL AND INGUINAL REGION

SERIES I

EXPERIMENT 1.—July 1, 1923, large adult dog.

Abdominal reposition was made of both testes through oblique incisions in both inguinal regions. The spermatic cord was freed and the internal ring enlarged on both sides. The gubernacula were severed, the peritoneal cavity was opened on both sides and the testes were pushed into the abdomen. On the left, the tunica vaginalis testis was removed; on the right side it was left intact. The wounds were closed in layers.

After the lapse of two months, this dog was kept with two adult female dogs for a considerable length of time without impregnating them.

December 28, 180 days later, castration was done. The wounds had healed well. The left testis was free in the peritoneal cavity; the right was ensheathed in omental adhesions. There was no interference with the vascular supply of either testis. Grossly both testes were markedly reduced in size. The tunica albuginea was thickened. Microscopically there was total loss of the germinal epithelium. Only Sertoli cells remained within the seminiferous tubules. In some of these, the nuclei were small and the cells gave the appearance of empty sacs. Many of the tubules were distorted. The interstitial cells appeared quite prominent.

EXPERIMENT 2.—July 5, 1923; an adult dog.

Abdominal reposition was made of both testes through inguinal incisions. The tunica vaginalis testis was left intact on both sides.

July 11, six days later, the dog was killed. There was a small hematoma in the right wound; the left was well healed. The right testis was free in the peritoneal cavity at the brim of the pelvis. The left testis was free except for slight attachment to the great omentum. Both testes were somewhat smaller than at operation.

Microscopically, there was definite loss of the germinal epithelium within the tubules. Numerous giant cells and débris occupied the lumina of the tubules. The interstitium was normal.

EXPERIMENT 3.—July 9, 1923; an adult dog.

Abdominal reposition was made of the right testis. The left testis was not touched.

July 14, five days later, castration was done. There was the same picture as in dog 2. The left testis showed normal spermatogenesis.

EXPERIMENT 4.—July 27, 1923; an adult dog.

Abdominal reposition was made of both testes through inguinal incisions.

August 6, ten days later, castration was done. The loss of germinal epithelium was a little more definite than in dog 2. Giant cells and necrobiotic changes were evident in all tubules. In many most of the sexual cells had disappeared. There was considerable peritubular edema.

EXPERIMENT 5.—Feb. 20, 1925; an adult dog.

Both testes were put into the peritoneal cavity through inguinal incisions.

April 13, fifty-two days later, castration was done. The testes were reduced in size; the tunica albuginea was thickened; the germinal epithelium was gone. Only a single row of Sertoli cells remained in the tubules. No products of necrobiosis were present. The interstitium was normal.

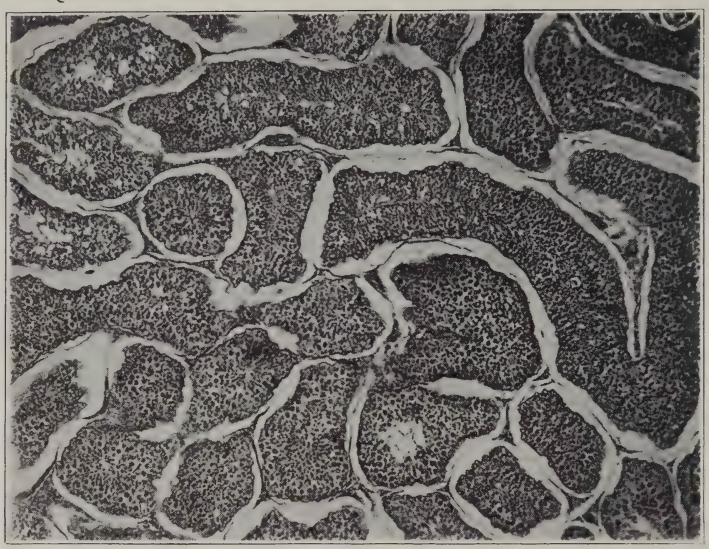

Fig. 1 (experiment 3).—Left testis of adult dog; control in scrotum showing the normal condition in the mature testis in which all the mature germinal cells are present (× 100).

Fig. 2.—Right testis of same dog as shown in figure 1 after having been in the peritoneal cavity for five days; marked destruction of the germinal epithelium; formation of multinucleated cells; Sertoli cells still in fair state of preservation; interstitium normal (× 100).

Experiment 6.—March, 17, 1925; an adult dog.

Through inguinal incisions the testis was removed from the scrotum on both sides after severing the gubernacula. The spermatic cords were mobilized and both testes were placed in the inguinal region beneath the fat of the abdominal wall.

May 13, fifty-seven days later, castration was done. The left testis had come to be just beneath the skin in a slight outpouching of the skin about 2 inches (5 cm.) distant from the scrotum. The right testis lay beneath the superficial fat. The left testis was larger than the right. The scrotum was shrunken on both sides. The microscopic examination was the same as in dog 5.

Young dogs were used in the following experiments:

Experiment 7.—Aug. 11, 1923; a pup, aged 4 months.

Both testes were placed in the peritoneal cavity in the usual manner.

Fig. 3 (experiment 8).—Right testis of dog approaching puberty that has been in the scrotum for ninety-one days; the large mononucleated cells in some of the lumina illustrate the first change noted in the young dog's testis (× 100).

August 18, seven days later, the dog was found dead. A typical normal pup's testis was found. A lumen was established in the tubules. There was definite arrangement of the Sertoli cells at the periphery with only scattered spermatogonia present. There were no giant cells, no débris in the lumina and no evidence of necrobiotic changes in the tubules. There was slight peritubular edema. (Shrinking in fixation may be partially responsible for this appearance.)

Experiment 8.—Sept. 19, 1923; a small pup, aged 3 months.

The right testis was placed in the peritoneal cavity, and the left testis allowed to remain in the scrotum for control.

December 19, ninety-one days later, the dog, which was mangy, was killed. The right testis was slightly adherent to the parietal peritoneum; the left was free in the scrotum. Both were about the same size.

Microscopically, the testes were those of a normal young pup. There was no elaboration of the germinal epithelium above the spermatogonia. In some of the lumina of the tubules of the right testis, which was placed in the peritoneal cavity, there were large cells with acidophilic cytoplasm. There were no multinucleated cells and no débris in the tubules. (These cells probably represent the earliest degenerative phase in the testes of the young)

Experiment 9.—Sept. 21, 1923; a small young dog, aged about 3 months.

Both testes were placed in the peritoneal cavity.

November 19, fifty-nine days later, the dog was killed. The left testis was adherent to the abdominal wall; the right was free.

Microscopically, the testis was that of a normal pup. There was no evidence of degenerative phenomena. There were numerous spermatogonia. The germinal epithelium was from two to three rows in thickness. There was no débris in the lumina. Some peritubular edema was noted (shrinking in fixation?).

Experiment 10.—Sept. 22, 1923; a young dog, aged about 5 months:

Both testes were placed in the peritoneal cavity.

October 5, thirteen days later, the dog was found dead.

Both testes were free in the peritoneal cavity. Microscopically, both these testes showed the picture mentioned in experiment 8, viz., large mononucleated cells with a reddish cytoplasm within the lumina. There were no multinucleated cells and no débris.

Experiment 11.—Sept. 24, 1923; a 5 months old dog.

Both testes were placed in the peritoneal cavity.

October 5, eleven days later, the dog was killed. The right testis was free in the peritoneal cavity; the left was ensheathed in adhesions of omentum. The epididymis was swollen. The surface of the testis was hemorrhagic. The vessels were slightly twisted.

Microscopically, the right was a normal pup's testis. No degenerative changes were present. The left had many tubules hyaline in appearance. Loss of ability of the cells in the tubules to take a stain was apparent in several areas. There were many polymorphonuclear cells in the interstitium. (This testis apparently was the victim of torsion of its vessels.)

Experiment 12.—Sept. 29, 1923; a small pup, aged about 4 months.

Both testes were placed in the peritoneal cavity.

December 1, sixty-three days later, the dog died. The right testis was slightly adherent to the greater omentum at the anterior abdominal wall. The left testis was free. There was no torsion of the vessels.

Microscopically, there were large mononucleated cells with acidophilic cytoplasm in the lumina (as in experiments 8 and 10). The outline of the tubules was well preserved. No other degenerative changes were present.

Experiment 13.—Oct. 11, 1923; a pup, aged 3 months.

Both testes were placed in the peritoneal cavity.

December 19, sixty-nine days later, the dog was killed. Both testes were free in the peritoneal cavity. There was no vessel constriction. The histologic report* was the same as in experiment 12, 8 and 10; that is, there were large

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* Many of these young dogs operated on in the later part of 1923 had mange. Though the testis of the young is not as sensitive to the effect of debilitating diseases as is the testis of the adult, nevertheless in the testis immediately before puberty slight degenerative changes are noted. Not unlikely, these are the result of the mange and malnutrition as much as the effect of transplantation. mononucleated cells with the tubule histologically otherwise normal.

Experiment 14.—Oct. 13, 1923; a small dog, aged 4 months.

Both testes were placed in the peritoneal cavity.

January 24, eighty days later, the dog died.

Microscopically, there was a many layered germinal epithelium in the tubules made up of spermatogonia. In a few tubules the mononucleated cells were present; an occasional multinucleated cell was found in some tubules. No loss of germinal epithelium was apparent. There was no débris in the lumina.

Experiment 15.—Dec. 23, 1923; a small pup, aged about 4 months.

Both testes were removed from the scrotum and placed beneath the skin in the peritoneum.

Jan. 21, 1924, twenty-five days later, the dog died.

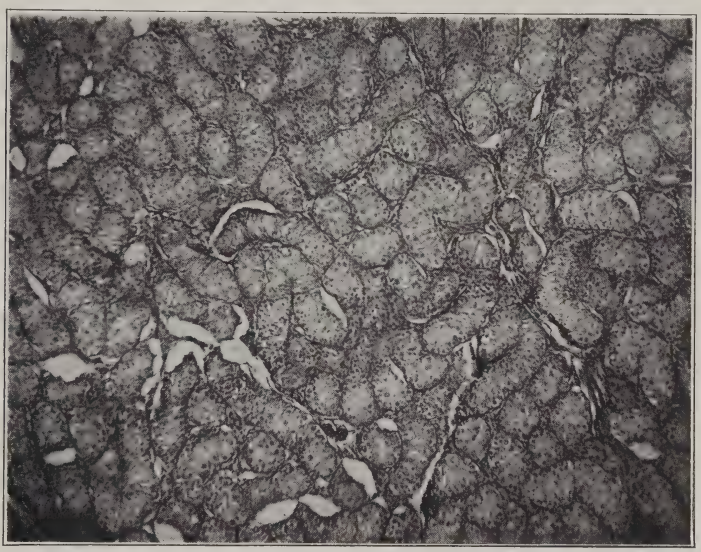

Fig. 4 (experiment 17).—Left testis of young dog; the control in the scrotum shows the normal condition of the young dog's or prepuberty testis (× 100).

Microscopically, a many rowed germinal epithelium was made up of spermatogonia. The contour of the tubules was well preserved. An occasional large mononucleated cell was present in some of the lumina. There was no débris in the lumina.

Experiment 16.—Feb. 27, 1925; a small dog, aged 3 months.

Both testes were placed in the peritoneal cavity.

March 15, seventeen days later, the dog died. The testes were adherent to one another in the peritoneal cavity.

Microscopically, there was a normal prepuberty testis. No degenerative phenomena were present. The germinal epithelium was from two to three layers in thickness. No adult germinal cells were present.

Experiment 17.—July 28, 1925; a good sized pup, aged about 5 months.

The right testis was placed in the peritoneal cavity. The left was not touched.

August 17, twenty days later, castration was done. Both testes were of the same size and were microscopically and histologically similar. A many rowed

germinal epithelium was present in both. No degenerative phenomena were present.

EXPERIMENT 18.—July 28, 1925; pup 4, aged 5 months.

The right testis was placed in the peritoneal cavity; the left was allowed to remain in the scrotum.

August 18, twenty-one days later, castration was done. Both testes were the same size. Microscopically, the germinal epithelium was from two to three layers in thickness. Both were histologically alike. No degenerative phenomena were present and no adult germinal cells.

EXPERIMENT 19.—July 31, 1925; a small pup, aged from 2.5 to 3 months.

The right testis was transplanted into the inguinal area; the left was not touched.

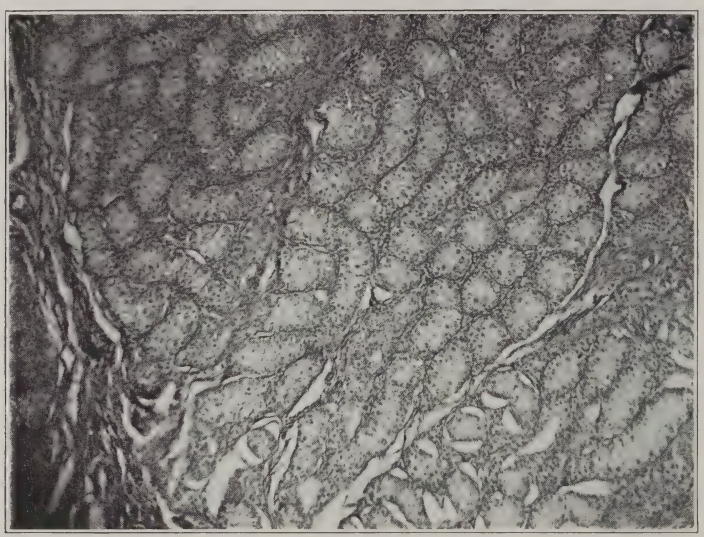

Fig. 5.—Right testis of same dog as shown in figure 4 after having been in the peritoneal cavity for twenty days; the only germinal cells present in either section are the spermatogonia; there is no appreciable histologic difference ($\times$ 100).

August 27, twenty-seven days later, castration was done. Both testes were the same size. Microscopically, a many layered germinal epithelium was present. No degenerative phenomena were observed. Histologically they were similar.

EXPERIMENT 20.—July 31, 1925; a small pup, aged 6 weeks.

The right testis was transplanted into the inguinal region; the left was not touched.

August 27, twenty-seven days later, castration was done. Both testes were of the same size. Microscopically, the central lumen was not established. There was a single layered germinal epithelium. The interstitium was prominent. Both were alike, and no degenerative changes were present.

The transplanted testis when removed, after a few days in its new environment, though appearing grossly much the same as its fellow in the dog's scrotum, microscopically shows a marked degeneration of the

germinal epithelium. This dissolution of the cellular elements constituting the walls of the seminiferous tubules is most striking. Heaped up in the lumina of the tubules is a cellular débris surrounded by an irregular and much thinned and scarcely recognizable germinal epithelium. In the lumina of many of these tubules are multinucleated or giant cells. The interstitium is not affected. After from two to three weeks have intervened following removal of the testis from the scrotum, the germinal epithelium has almost disappeared. Only here and there a few spermatogonia persist in the seminiferous tubules beside the Sertoli cells. The débris in the lumina disappears and after a few months absence from the scrotum the seminiferous tubules have the appearance of vacuolated rings with a single layer of cells constituted by the Sertoli cells.

The spermatogonia are the most resistant of the germinal cells and are the last to disappear. Maximow,[180] who has studied in an exhaustive treatise the histologic changes that occur in the testis after injury, states that the spermatogonia do not participate in the giant cell formation. The removal of the necrotic masses and giant cells from the lumina he ascribes to the phagocytic action of the Sertoli cells and connective tissue phagocytes and to dissolution and removal through the epididymis.

In the young dogs' testes in which spermatogenesis had not occurred none of these histologic changes were observed. No difference was noted between the control organ in the scrotum and the testis deposited in the abdominal cavity or inguinal region, in young pups from 2 to 3 months of age. However, in a few young dogs from 4 to 6 months of age, in whom spermatogenesis had not yet been established, a few large cells with acidophilic cytoplasm were found in the lumina of the tubules. In these dogs the spermatogonia are probably in preparation to elaborate the more mature germinal cells, and have just begun to exhibit the sensitivity that is so apparent after spermatogenesis has supervened.

The spermatogonia are the most resistant of the germinal cells and are the last to disappear. But that they do disappear is evident from microscopic examination of the testes that have long been removed from the scrotum. Spermatozoa were found in the epididymis by fresh smear in many of the dogs that were castrated a few days after the original procedure. This is in agreement with the finding of sperm in the ejaculations of persons who have recently been castrated. Impregnation of the ovum usually occurs in the fallopian tube, so that the spermatazoon apparently is immune to the changes that befall the other germinal cells when the testis is removed from the scrotum. The spermatocytes are the most vulnerable and these Maximow [180] says contribute largely to the formation of the giant cells. After the removal of the products of

necrobiosis from the lumina only the Sertoli cells remain within the tubules. Many of these have altered nuclei with only a scant amount of chromatin and lie on the membrana propria of the seminiferous tubules as empty irregular sacs.

None of these degenerative changes were observed in the interstitium. On the contrary, the interstitial cells appear prominent and increased in number. An actual hyperplasia seems to have occurred. Oslund [203] believes, however, that this increase is probably relative because of the disappearance of the germinal epithelium.

Fig. 6 (experiment 21).—Right testis of adult dog mobilized as for transplantation into the peritoneal cavity but immediately redeposited in the scrotum; removed twenty-five days later; the damage incident to the manipulation is apparent (× 100).

EXPERIMENTS TO DETERMINE THE REACTION OF THE TESTIS TO INJURY

SERIES II

Experiment 21.—Aug. 4, 1923; an adult dog.

Both testes were freed from the scrotum and prepared as for transplantation into the peritoneal cavity, but were immediately replaced in the scrotum.

August 29, twenty-five days later, the dog died without apparent cause. The wounds were well healed. The testes were slightly smaller than at operation. Microscopically, there was marked destruction of the germinal epithelium, most of which had disappeared. A few spermatogonia still remained. In the lumina of the tubules numerous giant cells and débris were present. In many tubules the

products of necrobiosis had been removed. The picture was similar to actual transplantation of the testis.

Experiment 22.—Aug. 22, 1923; a large pup, aged about 5 months.

Both testes were loosened up as in abdominal reposition; the left gubernaculum was not cut; the right was severed and the testis and cord were mobilized. Both testes were left in the scrotum.

September 6, fifteen days later, the dog died. There was a small stitch abscess in the left wound. Microscopically, the picture was that of a pup's testis. A two rowed germinal epithelium was present. No degenerative phenomena were noted.

Experiment 23.—Dec. 19, 1923; an adult dog.

Both testes were mobilized as for transplantation into the peritoneal cavity but were replaced in the scrotum immediately. The tunica vaginalis testis was removed on the right but left intact on the left.

Feb. 29, 1924, seventy-two days later, castration was done. The testes were normal microscopically. All the adult germinal cells including spermatozoa were present. The tunica vaginalis testis was removed on the right, without consequence.

Experiment 24.—Dec. 20, 1923; an adult dog.

Both testes were mobilized well into the retroperitoneal space and were replaced in the scrotum immediately.

January 19, thirty days later, the dog was castrated. Microscopically, there were numerous areas in which the germinal epithelium had almost disappeared. In a few tubules numerous spermatogonia were still present. An occasional giant cell was present in some of the tubules. (In this section apparently, the injury had occurred; the destructive process was about at an end and the regenerative changes were beginning.)

Experiment 25.—Dec. 20, 1923; an adult dog.

The testes and spermatic cord in both sides were well mobilized to the peritoneum; the testes were allowed to remain in the scrotum.

December 26, castration was done. Six days later microscopically the condition was the same as after transplantation in a six day testis, though less uniform and of a milder degree. In areas the destructive process was not much in evidence; in other areas practically all the germinal epithelium had disappeared and only giant cells and débris occupied the lumina of the tubules.

When the adult dog's testis is loosened from the scrotum and perpared as for abdominal reposition but replaced immediately in the scrotum, the same changes are observed within the lapse of a few days as occur in the case when testes are actually placed in the peritoneal cavity, though in slighter degree. The disappearance of the germinal epithelium is not so uniform but the giant cells are present and the lumina are filled with the débris of the germinal cells in the process of dissolution. Considerable peritubular edema also is present. This is the reaction of the young testis to trauma. No dissolution of the epithelial cells within the lumina is noted in young testes. In the testes of the new-born, this peritubular edema and even hemorrhage into the intertubular spaces are well known.

TRANSFERENCE OF THE TESTIS TO THE PERITONEAL CAVITY OR INGUINAL REGION WITH REPLACEMENT IN THE SCROTUM AT A LATER DATE, WHEN DEGENERATIVE CHANGES HAVE OCCURRED IN THE TRANSPLANTED ORGAN

SERIES III

EXPERIMENT 26.—July 8, 1923; an adult dog.

Both testes were transplanted into the inguinal region.

August 20, forty-two days later, the right testis was redeposited in the scrotum. The left testis was removed.

October 29, seventy-one days later, the right testis was removed. Microscopically, the left testis showed a typical transplant testis in which the germinal epithelium had practically disappeared. The products of necrobiosis had been

Fig. 7 (experiment 27).—Right testis of dog after having been in the peritoneal cavity for thirty-seven days (× 100).

removed. In the right testis definite attempt had been made to regenerate the germinal epithelium. In areas all the adult sex cells were present, including a few spermatozoa.

In those tubules in which the spermatogonia had all disappeared, no regeneration was apparent.

EXPERIMENT 27.—Feb. 18, 1925; a large adult bulldog.

Both testes were placed in the peritoneal cavity.

March 27, thirty-seven days later, the right testis was removed. The left testis was replaced in the scrotum. The spermatic cord was not under tension. The testis was sutured to the bottom of the scrotum, and the tunica vaginalis testis covering the testis was included in the sutures that united the inner layer of the scrotum.

November 4, 190 days later, the left testis, which was of good size, was removed. It was movable in the scrotum. Microscopically, the right testis was a

typical transplant testis. Nearly all the germinal epithelium had disappeared. A few spermatogonia still persisted in most tubules. The necrotic elements had been removed.

In the left testis the regeneration that had occurred was remarkable. Practically every tubule had the normal condition reestablished with all the mature germinal cells present, including numerous spermatozoa.

Experiment 28.—March 3, 1925; an adult dog.

Both testes were transplanted into the inguinal region.

April 6, thirty-four days later, the left testis was removed and the right testis was replaced in the scrotum.

August 19, 135 days later, the right testis, which was of good size, was freely movable in the scrotum. Microscopically, there was marked degeneration of the germinal epithelium in the left testis. In the right elaboration of all the mature

Fig. 8.—Left testis of same dog as shown in figure 7, also in the peritoneal cavity for thirty-seven days but replaced in the scrotum and removed 190 days later (× 100).

germinal cells in most of the tubules had occurred. Here and there was a vacant tubule. Spermatozoa were present in abundance.

Experiment 29.—March 6, 1925; an adult dog.

Both testes were disclosed into the inguinal region after the gubernacula were severed.

April 16, 101 days later, the left testis was removed and the right testis was replaced in the scrotum.

September 29, forty-two days later, the right testis was not in the scrotal pouch but was at the lower end of the inguinal canal. The right testis was removed.

Microscopically, the left testis was the picture of atrophy following transplantation. A few spermatogonia persisted. The atrophy was more complete in the right than in the left testis. No germinal cells remained.

Experiment 30.—Aug. 4, 1925; an adult dog.

Both testes were transplanted into the inguinal region.

August 18, fourteen days later, the left testis was removed and the right was replaced in the scrotum.

November 4, seventy-eight days later, the right testis was removed. It was high in the scrotum but movable. Microscopically, the left was degenerate with most of the germinal epithelium gone. There was elaboration of all the mature germinal cells in many tubules of the right. Quite a number of tubules, however, were vacant. There was no evidence of degenerative changes. (Lack of regeneration in many tubules were probably due to the high scrotal position of the testis.)

Experiment 31.—Aug. 19, 1925; an adult dog (old).

Both testes were transplanted into the inguinal areas.

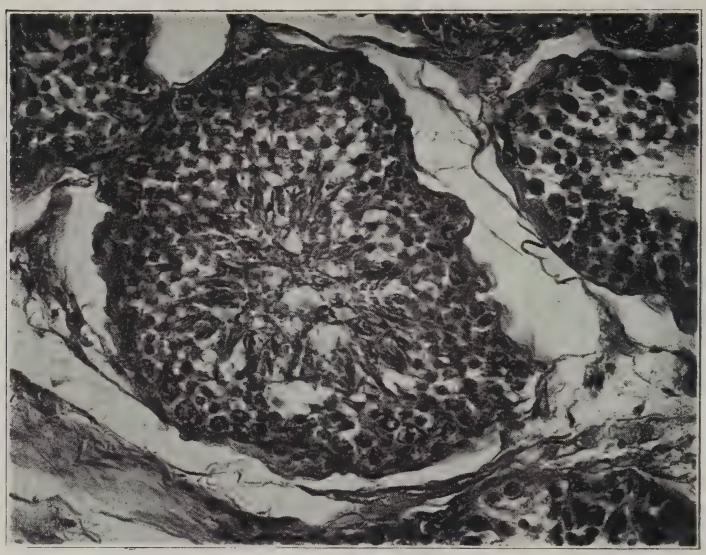

Fig. 9.—Same testis as shown in figure 8, under high power; figures 8 and 9 illustrate well the regeneration that occurs with replacement of the testis in the scrotum; all the mature germinal cells are present, and spermatozoa in abundance ($\times$ 250).

August 29, ten days later, the right testis was removed and the left was returned to the scrotum.

November 4, sixty-seven days later, the left testis was removed. The testis was movable in the scrotum. The degenerative phenomena were marked in the right testis and were still in progress. Giant cells and débris filled the lumina of most of the tubules. Regenerative phenomena were present in almost every tubule in the left testis. The germinal epithelium consisted largely of spermatogonia and spermatocytes. A few spermatids and spermatozoa were present.

Experiment 32.—Aug. 20, 1925; a large adult dog.

Both testes were transplanted into the inguinal areas.

September 2, thirteen days later, the left testis was removed and the right was replaced in the scrotum.

November 4, sixty-three days later, the right testis was removed. It was a large testis, freely movable in the scrotum. The left was a degenerate testis with necrobiosis still in progress. Giant cells were present in almost every tubule. Regenerative phenomena were quite uniform in the right. There was elaboration of all the mature germinal cells, including the spermatozoa.

Experiment 33.—Aug. 20, 1925; a large adult dog.

Both testes were transplanted into the inguinal region.

September 2, thirteen days later, the right testis was replaced in the scrotum. The left was removed.

December 15, 104 days later, the right testis was removed.

Microscopically, the left testis gave about the same histologic picture as the dog in experiment 32. Most of the germinal epithelial cells had disappeared.

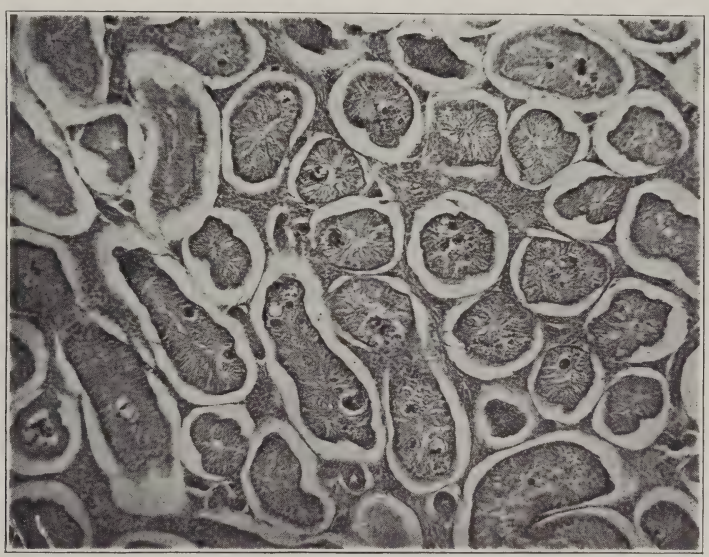

Fig. 10 (experiment 32).—Left testis of adult dog after having been in the inguinal region for thirteen days; the giant cells and débris have not yet been removed from the tubules.

Numerous giant cells were present. The normal condition had been reestablished in most of the tubules of the right. All the adult germinal cells, including spermatozoa, were present in abundance.

Experiment 34.—Aug. 21, 1925; a large mature dog.

Both testes were transplanted into the inguinal area.

September 4, fifteen days later, both testes were replaced in the scrotum.

December 15, 102 days later, both testes were removed. The scrotum was pendant on both sides; the testes were movable in the scrotum. Both testes were of good size.

Microscopically, both testes showed the normal adult condition present in nearly all the tubules.

Experiment 35.—Aug. 21, 1925; a large adult dog.

Both testes were transplanted into the inguinal area on each side.

September 10, twenty-one days later, the left testis was removed; the right testis was replaced in the scrotum.

December 15, ninety-six days later, the right testis was removed; a large testis was movable in a pendant scrotum.

Microscopically, there was almost complete disappearance of most of the germinal cells in the left testis. Only a few spermatogonia remained. In the right the normal condition had been reestablished in most of the tubules. In some there were no adult germinal cells.

EXPERIMENT 36.—Sept. 1, 1925; a medium sized adult dog.

The right testis was transplanted into the inguinal area. The left testis was allowed to remain in the scrotum.

September 17, sixteen days later, the right testis was replaced in the scrotum.

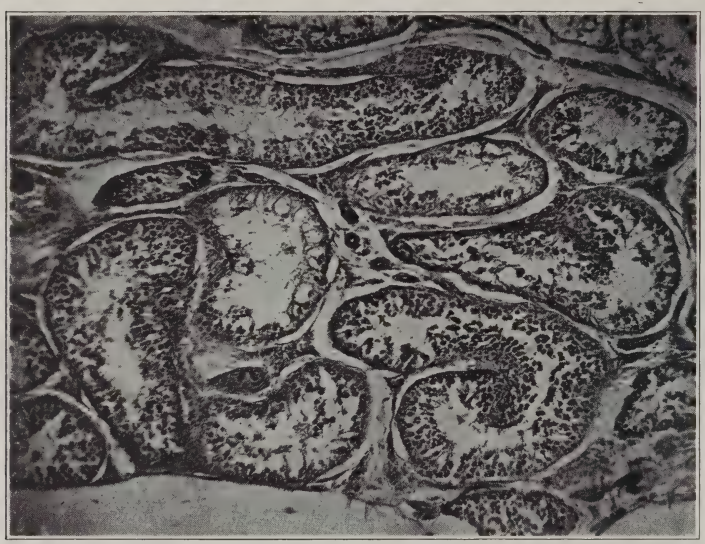

Fig. 11 (experiment 32).—Right testis of same dog as shown in figure 10, also in the inguinal region for thirteen days but replaced in the scrotum (when the left testis was removed) and allowed to remain in the scrotum for sixty-three days. Regeneration in a much shorter time is possible when the testis does not remain too long outside the scrotum.

December 18, ninety-two days later, both testes were removed. The testes were about of the same size, movable in the pendant scrotum.

Microscopically, the left was normal and there was elaboration of all the adult germinal cells in nearly all the tubules of the right.

EXPERIMENT 37.—Sept. 3, 1925; a large adult dog.

The right testis was transplanted into the inguinal region. The left was not touched.

September 16, thirteen days later, the right testis was replaced in the scrotum.

December 18, ninety-three days later, both testes were removed. The left was somewhat larger than the right.

Microscopically, the left was normal; marked regeneration was noted in most of the tubules of the right. A few showed little or no regeneration.

The testes of several mature dogs were transferred to the peritoneal cavity and inguinal region. After the lapse of a period of time, one testis was removed to determine the amount of injury and the other was replaced in the scrotum. These were subsequently removed and comparisons with the control organ made to determine whether regeneration of the germinal epithelium in the seminiferous tubules had occurred. In every instance but two marked regeneration of the germinal epithelium with formation of spermatozoa had occurred. The photomicrographs of the controls and the testis replaced in the scrotum illustrate well the ability of the germinal epithelium to be regenerated if the testis is redeposited in the scrotum. When the testis remains too long in the peritoneal cavity and most of the spermatogonia are destroyed the regenerative ability of the germinal epithelium is lost. This occurred in the fourth dog in this series. After remaining for 101 days in the peritoneal cavity, this testis was replaced in the scrotum. No evidence of regeneration was apparent after forty-two days. In the fifth dog in this series the testis had retracted out of the scrotal pouch and was found at the lower end of the inguinal canal. The physiologic position of the testis at the bottom of the scrotum must therefore be restored. In all these scrotal repositions, the testis was sutured to the bottom of the scrotum. The purse string suture used at the upper end of the scrotum prevents retraction out of the scrotal pouch, but this is not the proper location of the testis. This point will be discussed at greater length when the surgery of undescended testes is considered.

LIGATION OF THE VAS DEFERENS

SERIES IV

Ligation of the vas deferens was carried out on one side, and on the other all the vessels to the testis including the vessels accompanying the vas deferens were severed. Only the vas itself remained intact on this side.

EXPERIMENT 38.—Aug. 23, 1923; a pup, aged about 5 months.

The right vas deferens alone was ligated and severed on the left. All the vessels were cut and tied; only the vas itself was left intact.

September 7, fifteen days later, castration was done. There was only a small piece of scar tissue on the left. The right testis was of the same size as at the original procedure. Microscopically, the right showed a prepuberty testis in which no germinal cells beyond the spermatogonia had been formed. There were no degenerative changes. In the left only fibrous connective tissue remained.

EXPERIMENT 39.—Aug. 24, 1923; a large young dog.

The right vas deferens only was ligated and severed. On the left all the vessels to the testis and epididymis were severed and doubly ligated.

December 27, 125 days later, castration was done. Measurements of the right testis showed that it had increased in size since the previous procedure (to 4 by 3 by 2.5 cm. from the previous 3 by 2.3 by 2 cm.). Microscopically, in the right all the mature germinal cells were present, including spermatozoa. No sections were made of the left.

Experiment 40.—Aug. 27, 1923; a large mature dog.

The right vas deferens was ligated and severed. On the left all vessels were cut, leaving only the vas deferens intact.

December 20, 115 days later, castration was done. Only a small piece of scar tissue was noted on the left. The right testis was of good size. Microscopically, it was a normal testis with spermatozoa present in abundance. Only fibrous connective tissue remains in the left. Occasionally there was a suggestion of the outline of a previous tubule.

Experiment 41.—Sept. 4, 1923; a large adult dog.

The right vas deferens was ligated and severed. On the left only the vas deferens itself was allowed to remain intact.

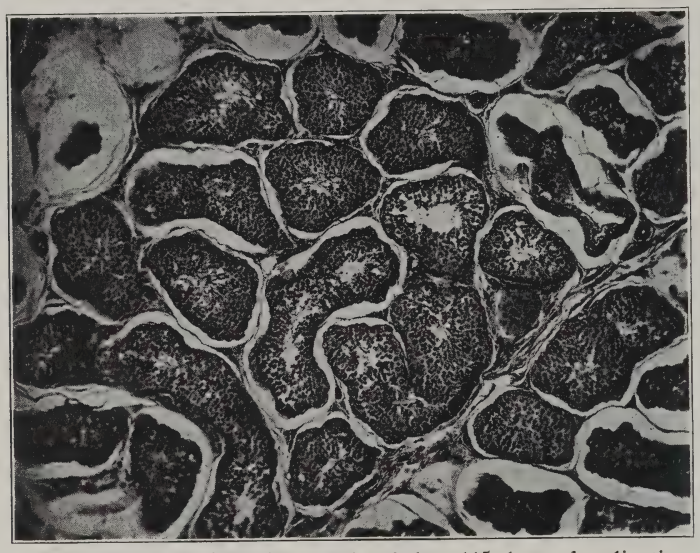

Fig. 12 (experiment 40).—Right testis of dog, 115 days after ligation of the vas deferens; the normal condition is present.

December 28, 15 days later, castration was done. There was only scar tissue on the left but a large testis on the right. Microscopically, the right was a normal testis. All the mature germinal cells and spermatozoa were present in all the tubules. No sections were made of the left.

Experiment 42.—Sept. 6, 1923; a large mature dog.

The right vas deferens was cut and ligated. On the left only the vas deferens itself was allowed to stay intact. All vessels were severed and doubly ligated.

September 24, eighteen days later, the dog died. There were gangrene and sloughing of the left testis. The right wound healed. Microscopically, on the right degenerative phenomena were present as observed in manipulation of the testis alone. No sections were made of the left testis.

Experiment 43.—Sept. 7, 1923; a large dog, aged 5 or 6 months.

The right vas deferens was doubly ligated and severed. On the left all the vessels were cut; only the vas deferens was left intact.

December 20, 102 days later, castration was done. The right testis had increased in size since the previous operation (from the previous 2 by 1.2 by 0.8 cm. to 3 by 2 by 1.5). Only a small piece of scar tissue was present on the left.

Microscopically, all the adult germinal cells except spermatozoa were in evidence. Numerous spermatocytes and spermatids were present. No sections of the left were made.

In these experiments no appreciable change was observed to occur in the testis following severance of continuity of the vas deferens when sufficient time was allowed to intervene before the dogs were castrated, to preclude damage attendant on the original operative procedure. Spermatogenesis continued despite the interruption of the excretory duct of the gland. No hyperplasia or hypertrophy of the interstitial cells was observed.

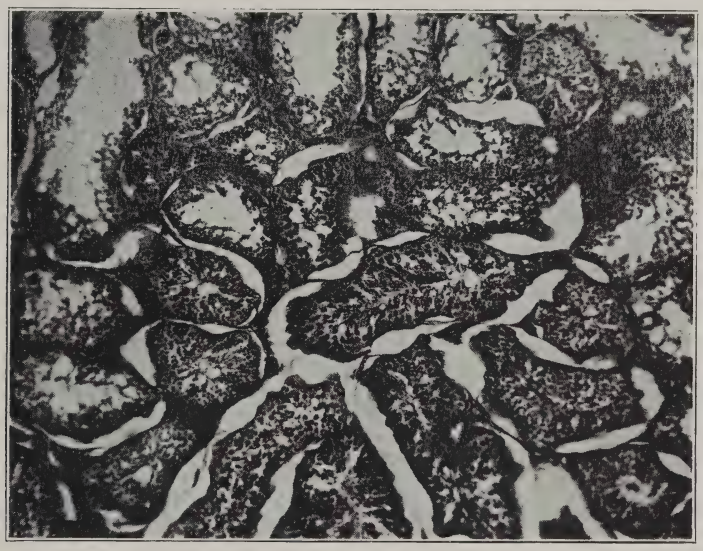

Fig. 13 (experiment 50).—Left testis of dog, 117 days after ligation of internal spermatic artery and anterior group of veins in the pampiniform plexus; the testis was much reduced in size, but the mature germinal cells were present. Ligation of the internal spermatic artery alone produces about the same picture.

BLOOD VESSEL LIGATIONS

SERIES V

EXPERIMENT 44.—Sept. 10, 1923; a large mature dog.

The internal spermatic artery and veins in the pampiniform plexus were severed and ligated on both sides leaving the vas deferens and its accompanying vessels intact.

December 28, 109 days later, castration was done. The dog had gained markedly in weight. The testes were small. Microscopically, no seminiferous tubules were recognizable. There was fibrous connective tissue replacement of the entire gland; numerous areas of pigment deposit were noted; the interstitium also was destroyed.

EXPERIMENT 45.—Sept. 11, 1923; a large mature dog.

The same procedure was used as in experiment 44.

December 28, 108 days later, the testes were small. The dog had increased in weight. Microscopically, the picture was the same as in experiment 44. The epididymis was normal. Only fibrous tissue and deposits of a brownish pigment persisted in the testis.

Experiment 46.—Sept. 17, 1923; an adult dog.

The vessels on both sides were ligated and severed, leaving the vas deferens and its accompanying vessels intact.

October 5, eighteen days later, both testes were large and injected. Microscopically, the contour of the tubules was still preserved. The lumina of the tubules was occupied by red blood cells. Most of the germinal cells had disappeared. There was marked edema in the interstitium. Most of the cells stained poorly.

Experiment 47.—Dec. 27, 1923; an adult dog.

The internal spermatic artery was ligated on both sides.

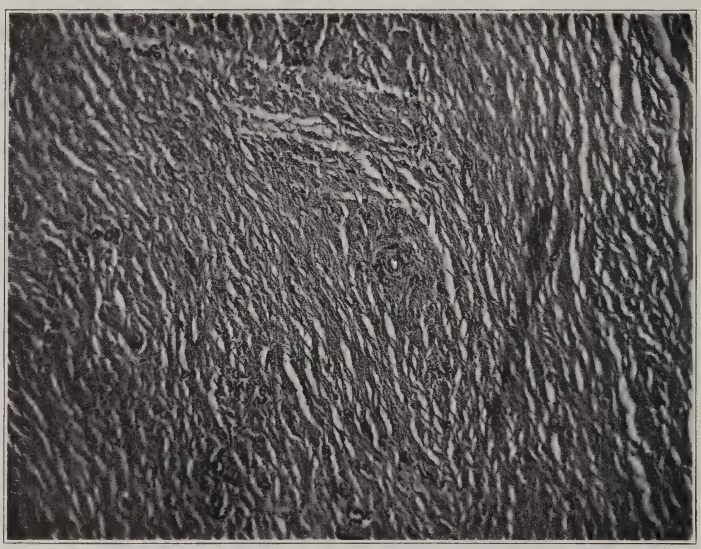

Fig. 14 (experiment 45).—Right testis of adult dog in which the internal spermatic artery and all the veins were ligated, leaving only the vessels accompanying the vas deferens intact; testis removed 108 days later; the parenchyma of the gland and interstitium as well were destroyed; only fibrous connective tissue remained.

Feb. 29, 1924, sixty-four days later, castration was done. Both testes were reduced in size. The microscopic section showed a fairly normal testis with all the mature germinal cells present. Only a few tubules at the periphery exhibited degenerative phenomena.

Experiment 48.—March 3, 1925; a large adult dog.

The left testis and spermatic cord were not touched. On the right the internal spermatic artery and anterior vein in the pampiniform plexus were cut and ligated, leaving the posterior vein and the vas deferens and the vessels accompanying it intact.

June 9, ninety-eight days later, castration was done. The left testis was about twice as large as the right. Microscopically the left was normal. On the right

there was a large area of fibrosis in the midportion of the testis. Most of the tubules showed normal spermatogenesis with spermatozoa present.

Experiment 49.—March 17, 1925; a large mature dog.

The internal spermatic artery was ligated and severed on both sides.

June 9, eighty-four days later, castration was done. Both testes were smaller than at the original procedure. Microscopically there was fibrosis in these areas. Spermatocytes and spermatids were present but no spermatozoa (a well elaborated germinal epithelium, however, which could develop normal spermatogenesis).

Experiment 50.—March 25, 1925; a large adult dog.

On the right the internal spermatic artery was ligated and severed. On the left the internal spermatic artery and anterior vein in the pampiniform plexus were ligated, leaving the posterior vein intact, as well as the vas deferens and its accompanying vessels.

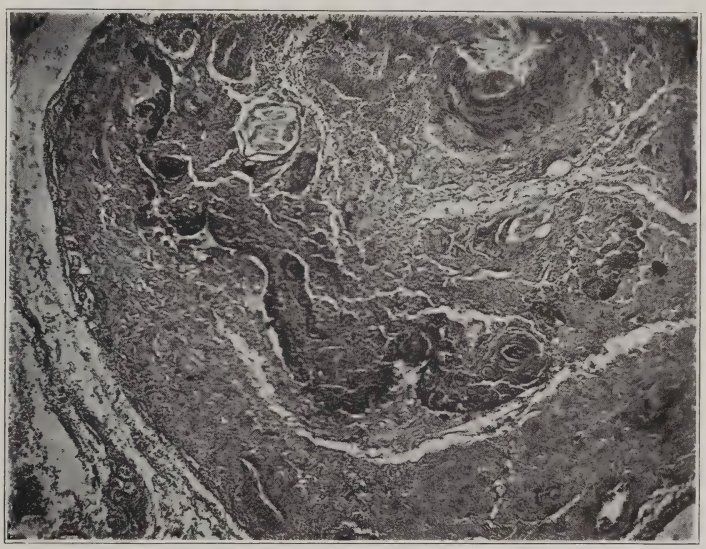

Fig. 15.—The testis of a boy of 19 who, six years previously, had an orchidopexy performed in which all the vessels, except those accompanying the vas, were divided, in order to place the testis in the scrotum; the similarity to figure 14 is apparent.

July 20, 117 days later, castration was done. Both testes were smaller. Microscopically, both appeared much alike. Spermatogenesis was present in most tubules. A few were empty. Numerous spermatozoa were present.

Experiment 51.—April 9, 1925; a mature dog.

On the right the veins in the pampiniform plexus only were ligated and severed. The internal spermatic artery and the vas deferens and its vessels were left intact. On the left the internal spermatic artery and the anterior vein in the pampiniform plexus were ligated, leaving the posterior vein and the vas deferens and its vessels intact.

July 20, 102 days later, castration was done. Both testes were smaller. On the right a fairly well differentiated germinal epithelium was present. There

were no spermatozoa. In many tubules at the periphery most of the cells had disappeared. (Separation of the vessels in the pampiniform plexus is always difficult because of the small size of the vessels. In all these experiments the internal spermatic artery, when cut, was allowed to spurt to be certain that the artery had really been severed. In the veins the procedure is not quite so simple. From what has happened in ligation of the internal spermatic artery alone and then ligation of the artery with one vein, and ligation of all the vessels save those accompanying the vas deferens, it is observed that only the latter procedure produces a complete degeneration. Separation of the vessels is attended with even greater difficulty just above the testis.) The left was fairly normal. There was elaboration of all the mature germinal cells. A few atrophic tubules were noted at the periphery.

Fig. 16.—Testis of a man of 22; three years previously this testis was high in the inguinal region; in order to place it in the scrotum, all the vessels except those accompanying the vas deferens were severed; the bare outline of a few tubules still persists.

Experiment 52.—Sept. 16, 1925; a large adult dog.

The internal spermatic artery and the anterior vein in the pampiniform were severed and ligated leaving the posterior vein intact on the right. On the left both veins in the pampiniform plexus were severed and ligated, leaving the internal spermatic artery intact. On both sides the vessels accompanying the vas deferens were not disturbed. The vessels in each instance were exposed in the upper portion of the inguinal canal.

December 15, ninety days later, both testes were removed. The right testis was considerably reduced in size. The left testis was about normal size.

Microscopically, on the right there were areas of fibrosis, especially at the periphery. Elaboration of most of the adult germinal cells in the remaining tubules was noted. The normal condition was present in most tubules of the left.

Invariably after ligation of all the vessels in the spermatic cord except the artery to the vas deferens, complete atrophy of the germinal epithelium and interstitium occur such that the parenchyma of the gland is replaced by fibrous tissue. The epididymis continues normal. In the animals in which castration was done after a shorter interval, necrosis of the parenchyma of the testis was observed. After ligation of the internal spermatic artery, a diminution in size of the testis is constantly noted. Many of these testes, however, appear fairly normal throughout the greater portion of the gland on microscopic examination. Here and there at the periphery atrophic tubules occur. A lesser number showed this partial degenerative process fairly well disseminated throughout the greater portion of the gland. Ligation of the internal spermatic artery with either the anterior or posterior group of veins in the pampiniform plexus leads to a rather more marked degenerative process than does ligation of the artery alone. When interruption of all the blood supply to the testis occurs, that is, ligation of the internal spermatic artery and veins in the pampiniform plexus together with the vessels accompanying the vas deferens, the testis shrinks up to a mere nubbin of scar tissue. Gangrene was observed only once and in this instance infection was present.

EXAMINATION OF UNDESCENDED TESTES IN MAN

SERIES VI

Seventeen testes that had failed to reach the scrotum were obtained for examination. Unfortunately no prepuberty undescended testes were available. In the testes of old subjects, even the seminiferous tubules had a hyaline appearance. The interstitial cells appeared increased in number. In two testes spermatogonia were observed. In the testis of a boy of 17 spermatocytes also were present. The similarity between the undescended testis in man and the experimentally ectopic testis of the dog is apparent.

THE TESTIS IN LARGE SCROTAL HERNIAS AND HYDROCELES

SERIES VII

Nine testes that were removed in the cure of large old scrotal hernias were examined. Fourteen testes were obtained for examination from patients who had had large hydroceles over a long period of time (8-12 ounces [236 to 355 cc.] of fluid present) and six were examined in whom both a scrotal hernia and a hydrocele had existed together.

As long as the testis continues in the scrotum, the elaboration of the adult germinal cells is possible. In the testes of men in whom large scrotal hernias or hydroceles had been present for years, the normal condition prevailed. The only anatomic peculiarity observed was an apparent increase in size of the lumen with a tendency to the formation

of diverticula and a thinning of the germinal epithelium. As all these testes were from men of advanced years, this finding is compatible with what has been described as the normal condition in old men.[253] In two men of 81, spermatozoa were observed. It is said that spermatogenesis disappears in the senile testis.[111]

EXAMINATION OF TESTES OF MEN DYING OF ACUTE AND CHRONIC FEBRILE DISEASES

SERIES VIII

Thirty-five testes were examined in this series. A scattered disappearance of the germinal epithelium appears in a number of testes of

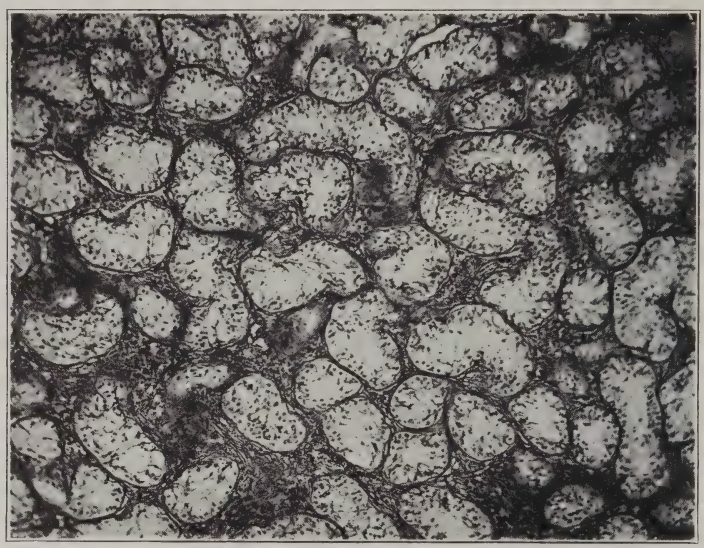

Fig. 17.—Inguinal testis of a boy of 17, removed during the course of a hernia operation; spermatogonia are in evidence; the more mature germinal cells, however, are absent; comparison should be made with a transplant testis.

persons dying of acute and chronic diseases. In a few the testis preserves the normal condition. The more uniform finding, however, appears to be a mild degenerative process in which the mature germinal cells disappear. Disappearance of the spermatogonia rarely occurs so that the normal condition may be quickly reestablished after the removal of the toxic agent.

COMMENT

The histologic change that occurs in the adult testis with removal from the scrotum has long been known. In 1891 Piana * interfered

* Piana: La Clinica Veterinaria, 1891, quoted by Stilling.[200]

with the descent of the testis in white rats and observed a marked atrophy of the germinal epithelium. Griffiths [112] in 1892 was the first to observe this change in dogs. He also noted the difference in reaction to abdominal transference of the testis in young and old dogs. In 1894 Stilling [260] placed the testes of twenty rabbits in the peritoneal cavity by severing the gubernaculum without even closing the internal ring; atrophy occurred in every instance. In 1905 Matsuoka [178] transplanted the testes of seven rabbits into the abdominal wall. At a subsequent date he cut the vessels and the vas deferens; a necrosis of the testis resulted. Schmidt [235] in 1913 confirmed Griffiths' [112] previous observa-

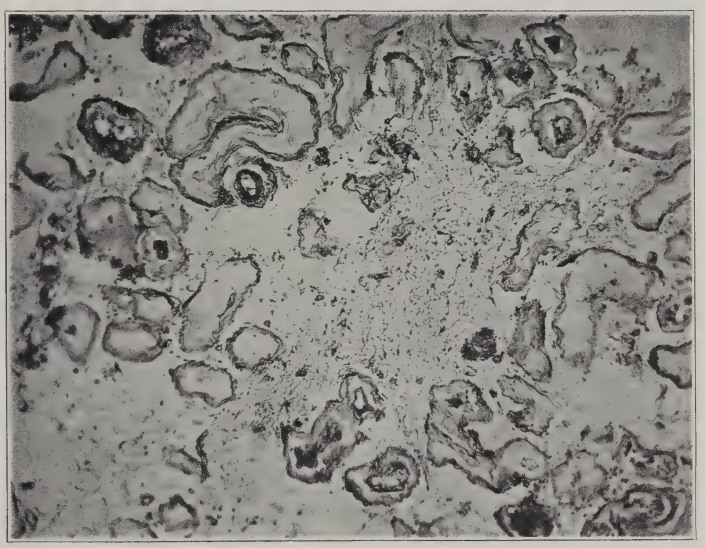

Fig. 18.—Inguinal testis of a man of 56 removed during the course of a hernia operation; no germinal epithelium is present; only a few Sertoli cells remain; the interstitium is normal.

tions. More recently Moore,[189, 190, 191] Fukui [102, 103, 104] and Fukiyiro [101] have noted the same effect.

Piana concluded from his experiments that the temperature in the abdomen was unfavorable for the testis.[260] Griffiths [112] could give no accurate answer as to the cause of the atrophy, but thought that the removal of the cremaster was responsible. Stilling [260] postulated that kinks in the epididymis were causative. Schmidt [235] believed that the lack of fixation of the testis transplanted into the abdomen and the abdominal pressure were responsible for its atrophy. Both Moore [190, 191, 193] and Fukui [103, 104] believe that the thermal factor is responsible for the degenerate condition of the testis removed from the scrotum.

Removal of the tunica vaginalis testis is only of transitory consequence. Alessandri,[4] Rolando,[224] Martini * and Schmidt [235] have made the same observation. Where removal of the tunica vaginalis alone has been done, the temporary degeneration that occurs is due to the manipulation, not to the deprivation from the testis of its superficial covering. Hanoaka [120] has shown that the tunica albuginea may also with impunity be removed. Only a few tubules at the periphery degenerate permanently.

Interference with the nerve supply of the testis has also been said to bring about degenerative changes in the testis. Kuntz [150, 151] and Takahashi [262, 263] have investigated this assertion of Obolensky [200] and have observed the same result. They believe that the sympathetic nerve exerts a trophic action on the testis. Lipschütz and Voss [168] in similar experiments on cats failed to observe atrophy in a single instance. Certainly in none of the above mentioned cases in which atrophy was observed was the factor of trauma satisfactorily eliminated.

The effect of ligation of the vas deferens on the testis is still a disputed matter. The Steinach theory of rejuvenation is founded [258] on the idea that atrophy of the germinal epithelium and hypertrophy of the interstitial cells follow occlusion of the vas deferens. Cooper [54] in 1823 ligated the vas deferens of a dog and six years later observed spermatozoa in the corresponding testis. Curling,[64] Gosselin [109] and Godard [106] were of the same opinion. Brissaud [32] failed to observe any histologic change in the testis following vas ligation, but postulated that this procedure put the testis in a resting state. Griffith [115] stated that vas ligation was without effect. Bouin and Ancel [27] in 1903 ligated the vas deferens in guinea-pigs and observed atrophy. At this time they suggested that the interstitial cells, which previously had been considered by most investigators as a trophic organ,[211] were responsible for the internal secretion of the testis. In young guinea-pigs they failed to observe the degeneration noted in the older animals. Recently they have repeated their earlier experiments on dogs [6] and obtained the same degenerative changes. Kuntz,[151] Van Wagenen [280] and Sand [229, 230] also find that occlusion of the vas deferens is followed by atrophy of the germinal epithelium. Fukui [104] states that vas ligation causes a degenerate testis. Martini † ligated and resected the epididymis at the caput, Steinach's site of election, but failed to produce other than a temporary atrophy of the germinal epithelium. Tiedje,[269] Retterer and Voronoff [221] find that the disappearance of the germinal cells is only transitory. Wheelon [291] failed to observe atrophy following vas ligation. Oslund [202] and Moore and Quick [192] state that no atrophy occurs following ligation

* Martini: Ztschr. f. Urol. 1908, pp. 728-745.

† Martini: Ztschr. f. Urol. 1908, pp. 728-745.

of the vas deferens. Oslund [202] calls attention to the fact that the testis must be known to be in the scrotum. In many cases, the ligation has been practiced on rodents in which the testes may be retracted out of the scrotum at will. Oslund [202, 203] and Moore and Quick [192] have not observed hypertrophy of the interstitial cells following vas ligations. In the experiments done by me the germinal epithelium did not disappear, nor was hypertrophy of the interstitium noted. When the testis is known to be in the scrotum and a sufficient length of time intervenes to preclude damage as a result of operative trauma, ligation of the vas deferens should be without event in normal healthy animals. Certainly

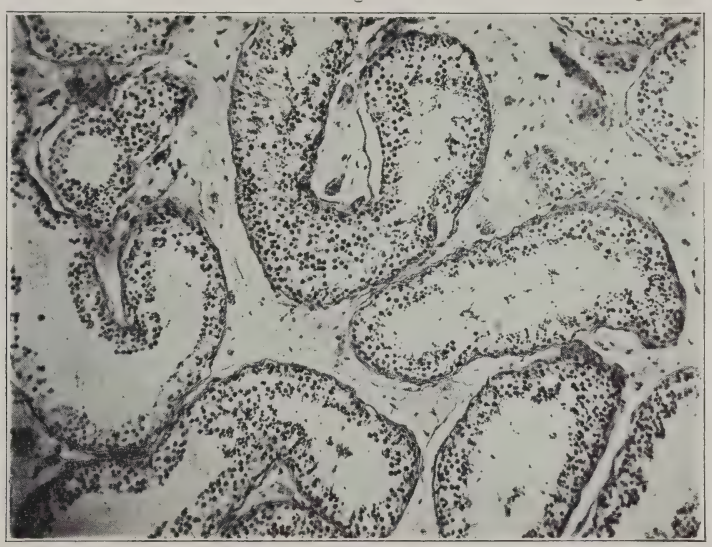

Fig. 19.—Scrotal testis of a man of 81 who had a large scrotal hernia and hydrocele for many years; the normal condition is present; the lumina appear unusually large and diverticula in the tubules are present—both normal findings in the testis of the aged.

any kinks that may occur in the vas deferens or epididymis when the testis is not in the scrotum is not the cause of the atrophy.

The rejuvenation of senile animals noted by Steinach [258] following this procedure is difficult to account for. In the results obtained by Steinach [258] and Sand [230] in patients the psychic effect or reaction to the operation may in a large measure discount the credit due the procedure. Certainly no histologic effect follows interruption of the vas in man or animals. In congenital absence of the vas deferens and after a number of years occlusion following gonorrheal epididymitis, the histology of the seminiferous tubules and interstitium still remain normal.[140]

Walker [285] and Fukui [104] have recently stated that the internal secretion arises in the spermatogenous elements. Though the internal secretion has not been definitely shown to take origin in the cells of Leydig, the weight of evidence would point toward these as its source.[118, 119] In cryptorchids in whom the secondary sex characters are preserved there is no germinal epithelium. Many of the Sertoli cells are in a degenerate state. Though the Sertoli cell cannot be ruled out of consideration as can the germinal cells, the available evidence would indicate that the interstitial cell is responsible for the internal secretion of the testis.

The difficulty of preserving as a graft so highly specialized a tissue as a testis is apparent. When all the vessels to the testis are ligated, a mere nubbin of scar tissue results. In such an experiment, an autograft is really created. Certainly the circumstances for a good result should be more favorable here than in a homo or hetero transplant. Lichtenstern [165] states that undoubtedly good results are obtained by homotransplantation of testicular grafts. Retterer's and Voronoff's [222] histologic studies of their own experiments on animals, however, would indicate the contrary.

In 1889 Brown-Sequard [36] demonstrated the efficacy of subcutaneous injections of testicular extract. Recently these results have been confirmed.[255] The administration of the active principle of the internal secretion of the testis by the hypodermic needle would appear to be more rational than the two procedures practiced today.

Neither is there any agreement as to the effect on the testis following the ligation of its blood vessels. Miflet [181] in 1879 held as a result of ligation of the internal spermatic artery in the dog that this vessel is an end artery and that its ligation causes atrophy of the testis. Examination of his protocols shows, however, that the degeneration in the germinal epithelium is only at the periphery of the tubules. Since then numerous anatomic researches on the blood supply * of the testis have been made in both man and dog. Janisch,[135] Colle,[48] Worms [297] and Picque and Worms [210] all state that the testis derives its arterial blood supply from three sources. The main source is through the internal spermatic artery which establishes anastomoses with the artery to the vas deferens and the external spermatic artery from the deep epigastric. No anastomotic flow occurs between the scrotal vessels and the arteries in the spermatic cord. After section of all the vessels to the testis, however, Colle [48] states that an anastomosis may obtain between the peripheral ends of the funiculoepididymospermatic system and the scrotal vessels sufficient to prevent gangrene of the testis when the usual source is interrupted. Pellanda and Neuhaus [199] fail to mention the external

* Enderlin [80] states that the testis will tolerate total occlusion of the blood supply for sixteen hours.

spermatic artery in their studies of the arterial circulation of the testis. Pellanda * also minimizes the importance of the deferential artery. Ludwig and Tomsa [172] state that the blood supply of the testis in the dog is the same as in man. Griffiths [113] finds that the vascular arrangement in man and dog are much the same. Schmidt [235] has studied the circulation of the testis in both man and dog and states that the comparison is not so complete as Ludwig and Tomsa [172] indicate. The source of supply is the same but the disposition of the vessels slightly different. The internal spermatic artery is more twisted in the dog and doesn't divide into two branches as soon as in the human being. Ellenberger and Baum [79] state that an anastomosis obtains in the dog between the three

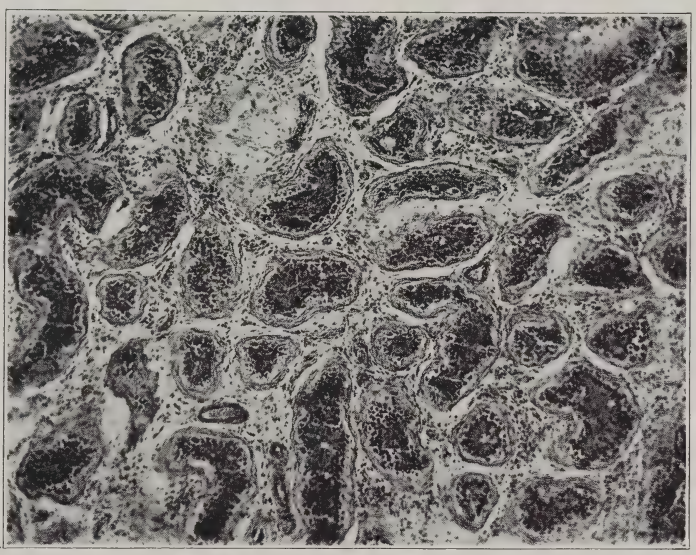

Fig. 20.—Testis of a man of 24 ill four years with pulmonary tuberculosis; no mature germinal cells are present; spermatogonia, however, are numerous.

vessels mentioned above. Experiments on the dog's testis are therefore applicable in the case of man.

In 1902 Mignon [182] in France advocated the section of all the vessels in the spermatic cord, with the exception of those accompanying the vas deferens, as a measure to aid low fixation of the testis in the scrotum in orchidopexy for incomplete descent. The following year, Bevan [17] independently in this country concluded from operations on varicocele in which he had sacrificed all the vessels in the spermatic cord except those accompanying the vas and in which no apparent interference with nutrition had occurred, that these vessels "could be safely sacrificed in

* Pellanda: Internat. Monatschr. f. Anat. u. Phys. **20**:240-265, 1903.

those cases where it is necessary to divide them in order to bring the testicle well down into the scrotum." Since then, this procedure has frequently been practiced with varying results.[136, 152, 184, 206, 290]

Previously Griffiths had demonstrated on dogs that ligation of all the vessels except those accompanying the vas deferens always results in atrophy. Griffiths [113] performed this experiment five times and only in one instance was the testis normal. But in that case the "ligature could not be found and free communication had become established as injections passed readily along the spermatic artery into the testis." In all the other instances cyanotic destruction, sloughing testis or extensive degeneration were noted. Griffith [113] did one experiment ligating the internal spermatic artery and the deferential artery; necrosis of the testis followed. In ligations of the internal spermatic artery alone, destructive changes were noted in the testes examined a few days after the ligation. But when a longer period of time intervened, the testis was found to be normal. Griffiths reasoned that the establishment of the normal histology was due to collateral circulation.

Alessandri [4] found that ligation of the spermatic artery and veins in the pampiniform plexus always caused degenerative changes. Ligation of the artery alone occasioned nutritive disturbances but no atrophy. Ligation of the artery to the vas deferens was without effect. Martini observed only a transitory effect on the testis after ligation of the internal spermatic artery.

Gangrene of the testis did not occur in experiments done by Moschowitz [196] in which the main bundle of vessels was ligated in dogs. Gessner [105] observed atrophy of the germinal epithelium of the testes in five dogs in which the same procedure was carried out. More recently Koyano [147] has practiced ligation in dogs of the vessels extraperitoneally internal to the anterior superior spine in order to eliminate the factor of trauma. In his hands, ligation of the internal spermatic vein provoke the least change in the germinal epithelium. Ligation of the internal spermatic artery and vein was followed by slight but more definite degenerative phenomena in the germinal epithelium. The greatest amount of atrophy Koyano [147] observed after ligation of the internal spermatic artery alone. He therefore believes that the procedure of vessel section in the treatment of undescended testis is rational and justifiable.

Haberer [117] has worked out in detail the venous outflow of the testis and epididymis. A great venous communication exists about the testis and epididymis Haberer states, and he finds that the blood may leave by four possible routes. Most of the return occurs through the internal spermatic vein, a lesser amount through the external spermatic vein, and portions through the vein of the epididymis and the vein of the vas deferens. The external spermatic vein empties into the deep epigastric;

the veins of the vas deferens into the plexus about the seminal vesicles, and the vein of the epididymis into the internal spermatic. Anastomoses between the veins, it is apparent, therefore, occur only in the lower portion. Interruption of the veins in the pampiniform plexus below the anastomoses will interfere considerably more with the circulation in the testis than the ligation of the internal spermatic vein alone high in the extraperitoneal region. This fact has long been known in the treatment of varicocele,[12, 131, 132, 146, 213] and the high ligation of the vessels has superseded the scrotal resection of veins. Koyano's observation, therefore, that ligation of the internal spermatic artery and vein in the

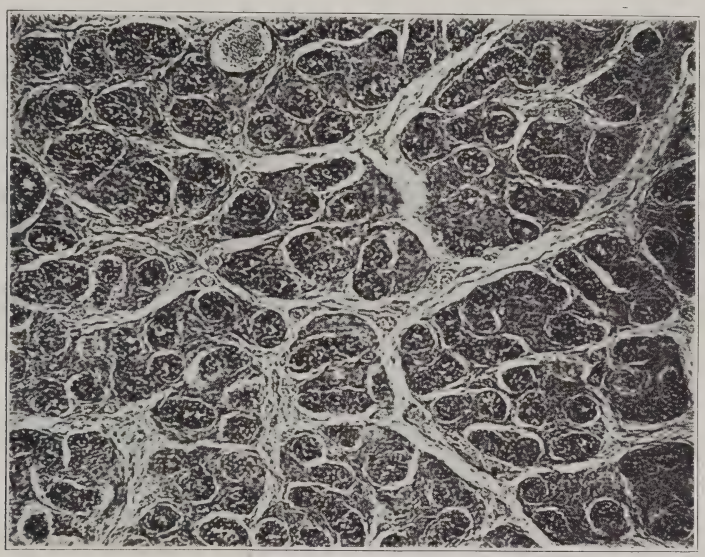

Fig. 21.—Testis of a seven months premature infant (one of triplets; the histologic picture was the same in all testes; the testes in each instance were still within the abdomen); the lumen has not been established in the tubules; Sertoli cells and spermatogonia alone constitute the walls of the seminiferous tubules; red blood cells are present in the intertubular spaces.

extraperitoneal region is without considerable effect on the histology of the testis is interesting, but amounts to virtually the same thing as my ligation of the internal spermatic artery and anterior group of veins in the pampiniform plexus; this ligation, as I have shown, may be compatible with a fairly normal spermatogenesis in the tubules. Marked diminution in size of the testis, however, occurs.

In the operation as practiced for scrotal fixation of the undescended testis, when vessel section is done, all the vessels except those in immediate relation with the vas are severed. Only rarely are the vas and the vessels that accompany it of insufficient length. The reaction of the

testis to division of the internal spermatic artery and vein in the retroperitoneal space do not therefore obtain in the operation as done for undescended testes.

Ligation of the internal spermatic artery and pampiniform plexus always results in atrophy of the germinal epithelium and interstitium of the testis as well. Statements [122] to the effect that ligation of these vessels is without deleterious effect on the testis will not bear investigation. A comparison of the photomicrograph that represents the testis recovered from a patient on whom orchidopexy with vessel section had been done a few years previously with what obtains in the dog is quite convincing as to what happens to the testis when all the vessels except those accompanying the vas deferens are divided.

Transference of the testis from the scrotum in adult dogs always results in atrophy. Whether the testis is placed in the retroperitoneal region, the peritoneal cavity or the inguinal region, degeneration of the germinal epithelium always occurs. Ligation of the vas does not cause atrophy. Ligation of the internal spermatic artery and with it the nerve supply to the testis does not cause a disseminated destruction of the seminiferous tubules.

When the testes are replaced in the scrotum, a fairly normal condition is reestablished. Spermatozoa are found in the tubules and in the epididymis. Moore [191] has recently also shown that regeneration of the germinal epithelium occurs in the seminiferous tubules of guinea-pigs and rabbits after scrotal replacement following elevation to the peritoneal cavity.

The scrotum, therefore, is essential to the normal development of a testis, and Moore [192] has rightly ascribed to the scrotum an important physiologic function.

Just what the scrotum affords the testis that is lacking elsewhere has been an interesting but mystifying problem. Piana concluded from his experiments that the temperature in the abdomen was unfavorable.[260] More recently Turner [275] examined the testes of perch during different seasons and found that with fall in temperature of the water spermatogenesis began and ceased with increase in temperature. Later Crew [61] pointed out that the scrotum was well equipped for local heat regulation in that no insulating layer of fat intervened between the skin and the dartos muscle; he suggested that this circumstances accounted for the aspermatic condition of cryptorchid testes. Benedict and Slack [11] had previously demonstrated that a constant body temperature is only reached at a depth of from 6 to 8 cm. and that a temperature gradient of 5 degrees obtains between the body cavities and the external surface. Fukui [102, 104] in Japan has recently observed that exposure of the dog's scrotum to the sun at temperatures of 44-49 C. for two hours produced degenerative changes in the germinal epithelium. The same occurred if the dog's

scrotum were immersed in water at the same temperature. The lowest temperature that will cause these changes he found to be 40 C. The higher temperatures required much less time to bring about the same effect. Moore and Quick [193] have observed these degenerative changes in the testis of guinea-pigs when exposed to increased temperature. Moore and Oslund [194] have also shown that following insulation of the scrotum of a ram with successive layers of woolen batting and cloth the same aspermatic condition is produced. Moore and Quick [193] observed a difference of 8 degrees C. between the temperature of the scrotum and abdominal cavity in guinea-pigs.

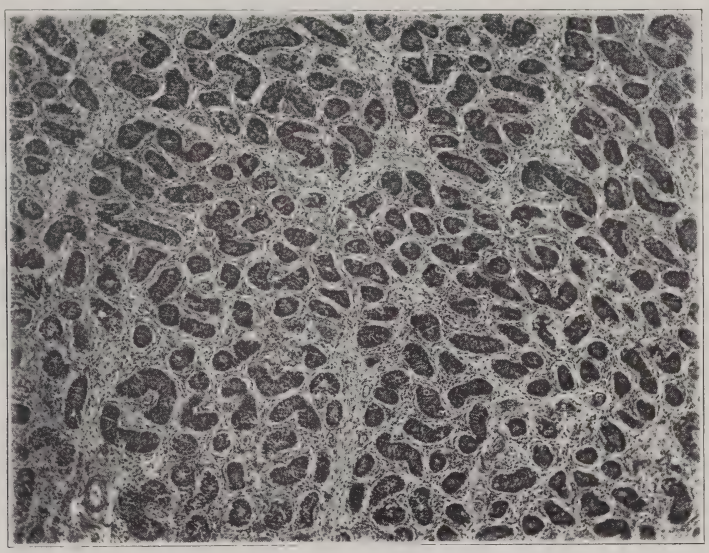

Fig. 22.—Testis of a 2 year old child.

These data would indicate that the scrotum exerts a beneficial influence on the testis through its ability to maintain a constant temperature. Whether the thermal explanation is correct or not, at least the scrotum is very necessary to the maintenance of a normal histologic picture in an otherwise normal healthy gland.

There are a few other facts that would support the heat factor as being of considerable significance. Hart [123] observed that elevation of the body temperature in gray mice when kept in iron cages at a temperature of 40 for five days causes atrophy of the germinal cells. It has long been known that in acute febrile diseases a degeneration of the germinal epithelium occurs. Stigler [259] showed that a temperature of 40.2 for four hours would cause the sperm to lose their motility.

The germinal epithelium apparently is sensitive to injury and a number of other agents can bring about its disappearance. Acute febrile

disturbances,[195] chronic debilitating diseases,[121, 145, 277] chronic alcoholism,[288] starvation [55, 170, 248] and vitamin free diets [5, 299] have all been known to bring about the same condition. Adler [2] reports injury to the seminiferous tubules after iodine administration. In one case coming under my observation this change was not noted.

In 1903 Albers-Schönberg [3] called attention to the sterility that occurs after exposure to the roentgen rays. Numerous observations [34, 35, 125, 148, 246] since then have corroborated this finding. As in the cases of starvation, acute and chronic febrile diseases, the removal of the cause enables the seminiferous tubules to be rehabilitated. So here the effect is usually transitory, unless the spermatogonia have been destroyed. In the prepuberty testis in which spermatogenesis has not been established

TABLE 1.—*Average Weight of Testis from Birth to Eighteen Years (Mita)*

	Gm.		Gm.		Gm.
New-born	0.5	1 year	0.7	2 years.....	1
5 years.....	1.16–1.23	11 years.....	1.93	12 years.....	1.9
13 years.....	4.50	15 years.....	11	17 years.....	16
18 years.....	17				

TABLE 2.—*Average Weight of Testis from Birth to Fifteen Years (Wwedensky)*

	Testis Weight in Grams		Epididymis Weight in Grams	
	Right	Left	Right	Left
New-born	0.2	0.196	0.12	0.12
3 months.....	0.52	0.51	0.19	0.19
1 year.....	0.72	0.71	0.2	0.2
1–5 years.....	0.86	0.87	0.19	0.2
8–10 years.....	0.8	0.82	0.24	0.24
11 years.....	1.2	1.3	0.4	0.39
12 years.....	1.5	1.5	0.42	0.42
14 years.....	1.48	1.5	0.52	0.52
15 years.....	6.79	6.83	1.00	1.00

this reaction is minimal.[294] No degenerative phenomena are in evidence, but edema and red blood cells may be present.*

Wwedensky [298] in an examination of forty-six cases from birth to fifteen years found the results given in table 2.

Elsewhere only brief mention has been made of the anatomy of the prepuberty testis. The general conformation of the testis as in the adult is ovoid, but bean shaped organs with a concave posterior aspect and a prominent hilum are not infrequent. Büdinger [39] has stressed this finding as significant in incompletely descended testes, but Gundobin [116] says this is not unusual in the normal. Mita [183] has weighed and microscopically examined 113 testes in ages from birth to 18 years. All but twenty-two of these were in children under 13. He appends a table of average weights (table 1), the amount being designated in grams or fraction thereof for one testis.

* Schultze [241] and Simmonds [244] state that this is a frequent finding in the apparently normal new-born.

Reich * has recently reported 426 testes measurements in the living by the use of calipers in 221 boys of various ages from birth to 16 years of age. A study of his measurements shows that the testis scarcely shows any definite appreciable growth before the eleventh year. Table 3 is a record of his measurements.

From these weights it is apparent that the testis increases but slowly in weight from birth to puberty. Scammon [232] states that the weight of the two testes at birth with their epididymides is about 2 Gm. During the first two years of life this weight is doubled, but until the thirteenth or fourteenth year the increase is very gradual, the average combined

TABLE 3.—*Measurements of Testis from Birth to Sixteen Years in 221 Boys (Reich)*

Age	No. of Cases	Left		Right	
		Length, Cm.	Breadth, Cm.	Length, Cm.	Breadth, Cm.
0– 1 month	6	1.5	0.7	1.6	0.8
1– 2 months	7	1.6	0.8	1.6	0.8
2– 3 months	11	1.6	0.8	1.6	0.8
3– 4 months	7	1.6	0.8	1.6	0.8
4– 5 months	4	1.7	0.8	1.7	0.8
5– 6 months	6	1.7	0.8	1.7	0.8
6– 7 months	4	1.6	0.8	1.6	0.8
7– 8 months	5	1.6	0.8	1.6	0.8
8– 9 months	5	1.6	0.8	1.6	0.8
9–10 months	4	1.6	0.8	1.6	0.8
10–11 months	5	1.6	0.8	1.6	0.8
11–12 months	7	1.6	0.9	1.6	0.9
1– 2 years	28	1.7	0.8	1.7	0.9
2– 3 years	13	1.6	0.9	1.6	0.8
3– 4 years	10	1.6	0.8	1.6	0.8
4– 5 years	5	1.6	0.8	1.6	0.8
5– 6 years	6	1.7	0.9	1.7	0.8
6– 7 years	14	1.7	0.9	1.7	0.9
7– 8 years	11	1.7	0.8	1.7	0.8
8– 9 years	13	1.5	0.9	1.6	0.8
9–10 years	8	1.7	0.9	1.7	0.9
10–11 years	11	1.7	0.9	1.7	0.9
11–12 years	8	1.9	0.9	2.0	1.0
12–13 years	7	2.3	1.2	2.3	1.2
13–14 years	6	2.8	1.4	2.8	1.4
14–15 years	6	2.8	1.4	2.9	1.5
15–16 years	4	3.5	2.0	3.6	2.0

weight of the testes and epididymides then being about 4 Gm. Mita's [183] figures show that the sudden increase in weight occurs about the thirteenth year. In Wwedensky's [298] statistics this marked increase is delayed until the fifteenth year. At all events, the increase in weight from the end of the first year to the time at which a sudden increase occurs is but a gram for each testis. Reich's data show that there is practically no increase in size of the testis until the beginning of the twelfth year, and Reich states that there is no growth of the testis during the first eleven years of life.

Spangaro [253] studied the histologic changes in testes from birth to old age and noted the similarity between those of the new-born and the prepuberty testes. Only with the increase of weight incident to puberty

* Reich: Testikelmessungen bei Kindern, Jahr. f. Kinderh. **105**:290-300, 1924.

and the occurrence of spermatogenesis does a marked change in the histology occur. In the testis of the new-born there is no real lumen in the tubules. Two or three irregular rows of cells almost completely fill the tubule. The spermatogonia may be present singly or in groups. Spangaro [253] states that the proportion of spermatogonia to Sertoli cells is from 1 : 35 to 1 : 200 to 1 : 400. In the preadolescent period the only changes that occur are the establishment of a central lumen; a definite arrangement of the Sertoli cells, and an increase in the number of spermatogonia, such that the proportion of spermatogonia to Sertoli cells increases to 1 spermatogone to every 12-20 Sertoli cells. However, Spangaro [253] states that a definite relationship between the number of

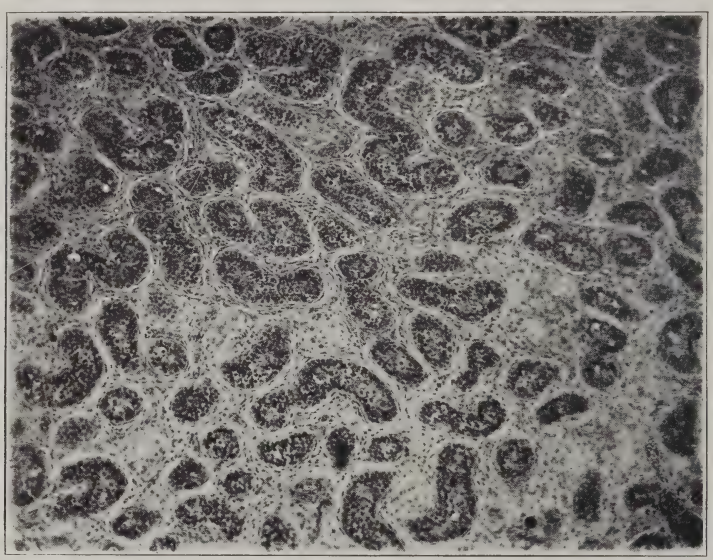

Fig. 23.—Testis of a 5 year old child; there is more definite outline of tubules than in figure 22 and beginning lumen formation.

spermatogonia in the tubules and the age of the child does not exist. The morphology of the spermatogonia themselves throughout the period from the first to the fourteenth year Spangaro finds is much the same as that of the new-born.

Mita [183] concurs in all of Spangaro's findings, but states that a rapid rate of growth occurs between the twelfth and the thirteenth years. Spermatogenesis was first observed at 13.

Wwedenski [298] classifies the changes that occur in the testis of the young in the following manner.

During the first fetal period from the fourth to the eighth month the tunica albuginea consists of but one layer and is of fine connective

tissue. Spermatogonia and Sertoli cells are present in the tubules. The average diameter of the tubules is 60 microns. The interstititial cells appear prominent and the connective tissue between the tubules is quite delicate.

In the second period from birth to 14 years, the tunica albuginea takes on a fibrous character and consists of two layers. The connective tissue between the tubules contains elastic fibers that do not become prominent, however, until 6 years of age. The epithelium of the tubules consists of nonfunctioning spermatogonia and the Sertoli cells.

During the third period beginning with the fifteenth year the tunica albuginea and corpus Highmori become thicker. The diameter of the

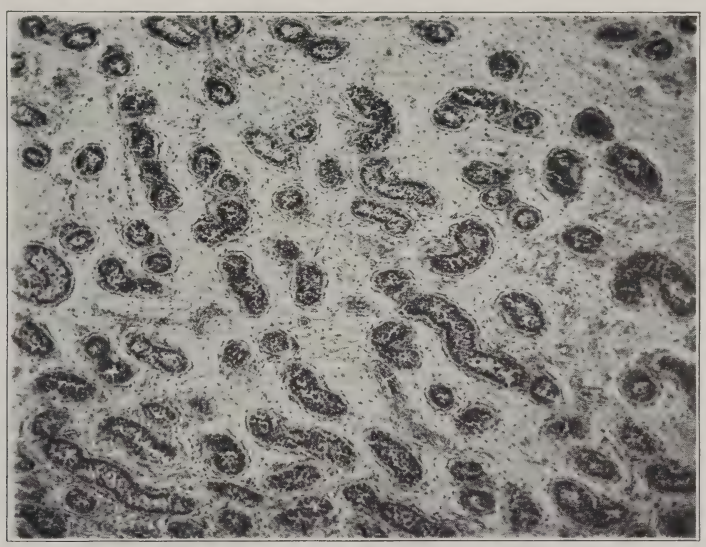

Fig. 24.—Testis of an 11 year old child; no mature germinal cells; increase in number of spermatogonia as compared with Sertoli cells.

tubules increases from 60 to 85-90 microns, and the adult germinal cells become differentiated.

It has previously been pointed out that the spermatogonia are the most resistant to injury and persist in the seminiferous tubules after transplantation of the testis from the scrotum for some time after all the other germinal cells have disappeared. In the prepuberty dog's testis in which only Sertoli cells and spermatogonia exist in the tubules, no change is observed in the histology of the organ when the gland is placed in the abdominal cavity or inguinal region under the same conditions that cause disappearance of the germinal epithelium in testes of the adult dog.

It is true that the spermatogonia in the mature dog's testis after a prolonged transference from the scrotum do disappear, and when replaced in the scrotum the mature germinal cells cannot be regenerated. When spermatogenesis supervenes the resistance shown by the resting spermatogone of the young animal is greatly diminished and it becomes as the other germinal cells, though in less degree, the victim of its new environment.

The ability of the testis to regenerate its structure has been the subject of a number of researches.[110, 133, 231] Anatomists are divided concerning the mode of origin of the germinal cells. One school believes that the Sertoli cells are the progenitors of the entire germinal epithelium,[214] the other that the Sertoli cells have only a nourishing function [10] and that the mature sexual cells are all developed from the spermatogonia that are present at birth. If the dog's testis is allowed to remain in the peritoneal cavity until all the spermatogonia have disappeared, no regeneration of the germinal epithelium is observed when the testis is replaced in the scrotum.

Before puberty the experimentally undescended testis develops much like its fellow in the scrotum. But if allowed to continue in its new habitat, it will make an attempt to maintain a normal spermatogenesis, but degenerative changes frustrate its attempts.

In how far can these data be construed to have any bearing on the human testis? It has been shown that no destructive changes are observed in the testes of young dogs when transferred to new surroundings outside the scrotum. It has been offered in explanation that the germinal cells in the testis at this time are few, the testis itself is small and only the onset of spermatogenesis makes the testis sensitive to injury. Transference of the testis in the adult dog from the scrotum always results in atrophy, but replacement in the scrotum at not too late a date restores the spermatogenic function again.

Frequent reference has been made throughout this article to Moore's excellent work on experimental cryptorchidism. Recently [191] he has shown that the testis of the rat and guinea-pig when placed in the peritoneal cavity will regenerate its germinal epithelium if redeposited in the scrotum. He concludes that this unquestionably demonstrates that the undescended testis is degenerate because of its position. The first experiments in this study were on guinea-pigs and rabbits. It occurred to me early, however, that in order to draw any conclusions relevant to the undescended testis in man an animal must be used in which the testis is always resident in the scrotum. The testes of guinea-pigs and rabbits, like those of all rodents, can be drawn up at will into the abdomen.[205] Tandler and Grosz [264] studied the seasonal changes in the testes of the mole. They found that during rest in the month of March the germinal epithelium reaches its highest development and then gradually wanes.

Rasmussen [215] and Hansemann [121] noted that the germinal epithelium disappears in the hibernating woodchuck. Marshall [176] says that in rodents the testes go back into the abdomen after rest, and that in testes of tame rabbits in winter there is little evidence of spermatogenesis. These reports have been confirmed by others.[59, 114, 166]

After finding in the abdominal transplantation of the testes in rabbits that the control left in the scrotum had also slipped into the abdomen, I abandoned rodents as the subject of experiment and chose the dog whose testis like that of man is continually resident in the scrotum. I am aware that Regaud [218] examined the testes of rats and found spermatogenesis present at all seasons of the year, but the scrotum is not the normal location of rodents' testes and I feel that it would be hazardous to apply to a testis whose habitat is always the scrotum the same standard that obtains in a testis that can be drawn into the abdomen at will. In the human being "ectopie en retour" always results in atrophy. In rodents this is physiologic.

A group of animals known as Testiconda,[205, 270] including the elephant, walrus, seal and camel, normally have a testis in the abdomen or inguinal region. In these animals this position of the testis is compatible with fertility. In the ram, boar,[9] the bull, horse [126, 127] or dog this position of the testis as in man is associated with an atrophic condition of the testis. Hobday [126] states that the nearer the testis approaches the scrotum in horses, the more likely is that testis to exhibit spermatozoa.

Numerous authors state that the anatomy of the human undescended testis is normal before puberty.[20, 161, 174, 252] Unfortunately only few of these have been obtained for microscopic examination. However, the examination of a small number of these have been recorded and in practically every instance they have been described as normal. Felizet and Branca,[86, 87, 88, 89, 93] to whom we are indebted for a number of these examinations, though believing that a primary atrophy of infancy and a secondary postpuberty deterioration bring about the degenerate condition of the undescended testis, nevertheless state that the phenomena of degeneration are much less frequent in the undescended testis of the young subject than in the adult. Their examinations of undescended testes in young subjects show that a germinal epithelium represented by spermatogonia, spermatocytes and spermatids is elaborated.[86, 89] They believe that atrophy occurs before spermatozoa are formed. A testis that can produce spermatocytes can also produce spermatozoa and maintain a normal spermatogenesis under the proper environment as observed in scrotal replacement of abdominal testes in dogs.

Felizet and Branca [86, 89] in two articles on the histology of the undescended testis in the young record the examination of twenty-four undescended testes in their first paper, fourteen of which were in the

prepuberty or puberty testes. In their following article they report twenty-six cases; eleven of which were in the young. Excerpts from the descriptions of the testes in their second publication on the subject are tabulated. Their conclusions and findings were the same in both articles.

Of the fourteen undescended testes examined in the first series, eight were said to be normal for the age. (Included among these, eight were testes of subjects from 11 to 14 years.) One in a boy of four was tuberculous. Of the remaining five, all occurred in subjects 13 years of age and the description given these was that the germinal epithelium was not commensurate with what one should expect at this age. From the researches of Spangaro,[253] Wwedenski[298] and Mita,[183] however, it is apparent that the histology of the new-born child's testis and that of the boy of 13 before spermatogenesis occurs are very similar.

Tabulation of descriptions of the young testes in Felizet and Branca's second series follows:

1. Observation 29. Age, 6. Spermatogonia in repose; some of these in mitosis.
2. Observation 33. Age, 9. The seminal tubules were constituted by epithelial cells and a number of male ovules (spermatogonia). "The canals are full but rare." The diameter of the canals varied from 26 to 45 microns. Only a maximum of thirteen seminal canals could be counted in any one fixed field with a number 4 objective. (In a footnote at the bottom of the same page they state that as many as from twenty-five to thirty tubules have been counted in another undescended testis in a boy of 9.)
3. Observation 34. Age, 15. Numerous spermatogonia in mitoses; degenerative changes also apparent.
4. Observation 36. Age, 14.5. Numerous spermatogonia, some in repose, others in mitosis; also spermatocytes.
5. Observation 39. Age, 17. Spermatogonia and spermatocytes; also degenerative changes.
6. Observation 40. Age, 8. Tuberculous testis.
7. Observation 42. Age, 6. Spermatogonia present, as many as five per tubule; lumen present in most of the tubules; in places desquamated elements from "male ovules" and Sertoli cells. (The latter statement is certainly open to doubt.)
8. Observation 44. Age, 12.5. Numerous spermatogonia in repose; many in process of division; also spermatocytes of first order; a number of Sertoli cells were in the process of degenerating and contained fat.
9. Observation 45. Age, 12. Some of cells were filled with fat; some were desquamating into the central lumen; did not stain as well as normally.
10. Observation 46. Age, 14. Chromatolysis of some spermatogonia; others in the process of proliferation; a few spermatocytes were present in several stages of mitosis.
11. Observation 47. Age, 14. A few areas of degeneration; some infantile tubules present; some showed vacuolated spaces from which the germinal cells were disappearing; some tubules were of normal diameter and showed spermatogonia and spermatocytes in mitosis.
12. Observation 50. Age, 14. Spermatogonia and numerous spermatocytes; also evidences of degeneration.

Bezancon mentions the examination of two undescended testes in the young. One of these was an inguinal testis removed from a boy of 13. When compared with a scrotal testis of a boy of the same age removed at postmortem, Bezancon states that there was no difference and that the histology of the undescended testis was normal. He cites one other case, that of a boy of 1 year whose undescended testis was smaller than the scrotal testis, but here there was also an abnormal blood supply in that the spermatic artery arose from the aorta just above its bifurcation.

Staemmler reports the examination of three prepuberty undescended testes:

1. Left inguinal testis, age, 15; weight of descended organ, 7 Gm.; weight of undescended organ, 3 Gm.; degenerative phenomena present in the undescended testis.
2. Right inguinal testis, age 6; same weight and appearance as the descended testis.
3. Bilateral inguinal testes, 4¾ years; "Kyrle's undeveloped testes."

A word of explanation here is necessary as to what the so-called underdeveloped testis of Kyrle [154, 155, 156, 157] is. Kyrle examined 110 testes in children. Eighty-six of these he found to be grossly hypoplastic and of the twenty-four, one-half were microscopically hypoplastic. Diamantopoulos [67] concurs in Kyrle's observation and finds that a normal testis in the young is a rare occurrence. Spangaro,[253] Mita [183] and Wwedenski's [298] descriptions of the normal condition in the prepuberty testis would agree with what Kyrle and Diamantopoulos describe as a hypoplastic gland. Reich states that a testis is only to be described as infantile if it fails to assume the normal growth after the eleventh year. Schultze's [241] findings are in agreement with those of Spangaro, Mita and Wwedenski. Voss,[284] using Kyrle's criteria, was able to find only seven normal testes in 117 examinations of children under 11. In thirteen boys from 12 to 20, however, the normal condition was observed in twelve, or 93.8 per cent. Voss makes the interesting comment that a hypoplastic child's testis can apparently assume the normal condition at puberty. The primary atrophy of undescended testes that Felizet and Branca [86, 89] recognize in infancy is apparently the so-called Kyrle's underdeveloped or hypoplastic testis, a normal finding in prepuberty testes.

The surgeon who palpates the undescended testis of the child and pronounces it atrophic commits the same error, not taking cognizance of the fact that the scrotal testis until spermatogenesis supervenes is also to palpation an "atrophic" organ.

There are a few cases on record in which sperm production or children have followed marriage after bilateral orchidopexy in the father. In

all these instances, however, the operation was done after puberty * and no record was obtained as to the absence or presence of spermatozoa before the operation. Because of the age at which the operation was done, I feel that these must be regarded as instances of those cases in which a temporary spermatogenesis may occur in cryptorchids.

All these facts do, however, demonstrate that the undescended testis goes through the state in which a mature germinal epithelium is elaborated. The very fact that a certain number of cryptorchids' testes are functional in an abnormal habitat demonstrates that they are not ab ovo imperfect. The fact that degenerative changes appear, as Felizet and Branca [86, 89] have described, in the undescended testis at puberty is significant that the more mature germinal cells are produced, for the immature or prepuberty testis does not exhibit degenerative changes. Similarly, the testis of the young dog undergoes no change when elevated into the inguinal region or peritoneal cavity. The adult dog's testis is subject to degenerative changes if removed from the scrotum, but when replaced in its normal environment before all the spermatogonia have disappeared, the histology of the tubules can be restored.

Given the stimulus to development, therefore, that the scrotum affords, the undescended testis of man, if successfully placed in the scrotum before the changes incident to puberty occur, should develop normally.

THE SURGERY OF THE UNDESCENDED TESTIS

No attempt will be made here to discuss the numerous procedures that have been suggested to deal with the undescended testis. The principles involved in the treatment of the condition will be briefly considered.

In general there are three methods practiced in the treatment of arrest of descent, viz., removal of the testis, abdominal reposition and orchidopexy. Szymankowski,[261] himself [16] the victim of malignancy in an inguinal testis, was a strong advocate of its therapeutic removal. Many support him in this belief.[267] Others urge removal of the inguinal testis only in the event of pain or other complicating factor, and still others believe that the inguinal testis should be removed [7, 173] after puberty if the other testis is in the scrotum. The testis within the abdomen has usually been left alone unless a hernia has necessitated

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* Duchesne [68] reports the case of a boy with bilateral incomplete descent. At 16 an inguinal testis was placed in the scrotum; at 17 a right inguinal testis was similarly treated, but retracted to the external ring. At 18 spermatozoa were found in the semen. Froehlich [100] reports the same finding in a boy of 18 who had been operated on the year before for bilateral inguinal retention. Coley [61] observed spermatozoa in the ejaculation of a man of 30 after the bilateral operation. Coudray [58] observed a boy whose testes descended spontaneously after 11 years of age; nine years later sperm were present.

interference. In bilateral abdominal retention, operation has been urged to preserve the secondary sexual characters under the impression that cryptorchids were not infrequently like castrates. From what has gone before, it is obvious that the ideal treatment of partial descent is fixation of the testis in the scrotum without injury to the vessels.

Age.—The best age at which to place the testis in the scrotum also is a matter of dispute. Most writers are agreed that the operation should be done before puberty. Brocha [33] states that the operation may be performed as soon as the child is diaper broke. Mixter [185] and Eisendrath [78] believe that the operation should be done at any early age. Eisendrath, however, doesn't believe that placing the testis in the scrotum favorably affects the spermatogenetic power of such a testis. Le Joly-Senoville [161] believes that from 14 to 16 is the proper age; Duchesne,[68] from 10 to 20, and Carlier,[44] from 17 to 25. The belief that pituitary [75] or thyroid [198] extract will encourage spontaneous descent is without foundation.

A consideration of the anatomy of the prepuberty testis and the reaction of the young dog's testis to transplantation outside the scrotum, however, indicate that the operation must be done before puberty changes supervene. Wwedenski [298] believes that 15 is the age of puberty. Spangaro [253] states that independent of race and individual difference the puberty age lies usually between the thirteenth and fifteenth years. Mita [183] first observed spermatogenesis in a testis of a boy of 13. Le Prince,[164] in a study of the time at which spermatogenesis occurs, first observed the presence of spermatozoa at 13.5 years. Crampton [60] studied the physiologic age in 48,000 New York high school boys and determined from the appearance of pubescence in these children that only 6 per cent were mature at the age of 12.5-13 years. Between 14.5 and 15 years, 60 per cent were mature; at 17.5-18 years, 100 per cent had reached the mature physiologic age.

Scammon [233] believes that even in the absence of the appearance of spermatogenesis in the young testis the changes incident to puberty may begin to occur as early as the ninth year. The reposition of the testes of young pups of from 4 to 6 months of age would also support this opinion. In the elevated testes of these dogs, though the degenerative processes observed in adult dogs did not occur, occasionally large mononucleated cells with acidophilic cytoplasm made their appearance. These probably indicate that the spermatogonia are preparing for the production of the mature germinal cells. In the testes of pups of from 2 to 3 months of age, these changes are constantly absent. As soon as spermatogenesis occurs in the young dog's testis, it becomes just as sensitive to removal from the scrotum as does the testis of the mature dog.

The undescended testis, therefore, should be in the scrotum before spermatogenesis occurs. Because of the occasional occurrence of late descent, the operation may well be delayed until the ninth or tenth year; not infrequently a testis that could not be palpated will come down into the canal during the first few years of life. The presence of a hernia may urge the performance of the operation at a relatively early date. It has previously been stated that the testis increases little in size until puberty. The increase in the musculature of the vas and cremaster parallels [209] the increase in size of the testis. He who advocates waiting [44, 245] until the structures are larger would jeopardize the function of the testis. I feel that the age of choice in which to do the procedure lies between the ninth and eleventh years. Only rarely will the placing of a testis into the scrotum as late as the sixteenth year be of any functional benefit. In the patient who presents himself for operation at a late date, the result can only be cosmetic.

OPERATIVE PROCEDURE

The modern operation of orchidopexy begins with Schüller,[240] It had been previously performed but with little success.[1, 46, 207, 226] Bevan's [16, 17, 18] admirable description of the procedure contains practically everything that is fundamental in the technic. Adherents of the procedure everywhere have since found that the separation of the testis from the vaginal process, the freeing of the vessels and vas deferens into the abdominal cavity, and the separation of the fascial coverings of the cord permit the mobilization of the testis. The suture of the lower portion of the vaginal tunic around the testis is not necessary to the success of the procedure.

In one particular, however, I cannot agree with Bevan. In many instances, as Bevan states, after the testis has been mobilized it may with ease be placed on the thigh several inches below the inguinal ligament. If the testis is then placed in the scrotum, one is surprised to find that in a few days it has retracted to the top of the scrotum. Bevan's purse string suture, it is true, will prevent the testis from being retracted out of the upper part of the scrotum, but this is not the physiologic position of the testis. Many believe that anchoring the testis at the radix peni accomplishes what nature had intended. However, such is not the case. Since Fukui's [102, 103, 104] and Moore's [191, 193] researches, it is apparent that the bottom of the scrotum is the psysiologic position of the testis. Under the influence of cold the scrotum retracts, and on warm days the scrotum is quite pendant and the normally situated testis follows the movements of the scrotum.

I therefore believe that suture of the testis to the bottom of the scrotum is necessary, but not this alone. A suture should be run through

the bottom of the scrotum and the testis maintained in its physiologic position for days under tension by mechanical fixation. *

It seems strange that the testis stripped of the cremaster and all the fascial coverings should retract. The number of poor results following orchidopexy, however, indicate that its does occur. When the mobilized gland hangs freely suspended by the vas and vessels alone, retraction signifies that one or the other has brought about the elevation of the gland.** Fecher [85] has investigated the elasticity of the vas and spermatic vessels and finds that the vessels will permit of twice as much stretching as the vas. It is physiologic for blood vessels to retract. Fecher, however, concludes that the vas is the cause of the retraction. I should infer from his experiments that the vessels being twice as elastic as the vas are much more likely to be the factor in the retraction. The greater ease with which the testis is maintained at the bottom of the scrotum after vessel section would also support this view.

In addition to lengthening of the spermatic cord by a method such as Bevan's, it has been suggested that the path the cord has to traverse can be shortened. Procedures such as those of Davison,† Wolfer [296] and Frangenheim [96] are directed toward this end. The chief increase in length that occurs, however, after division of the deep epigastric vessels or transplantation of the testis behind them is in the vas deferens and this structure is practically always long enough. The gain in length of the spermatic vessels by the maneuver is slight. Sievers [247] has suggested passing the testis through the obturator foramen.

If the internal spermatic artery and veins in the pampiniform plexus have to be divided to place the testis in the scrotum, atrophy of the germinal epithelium not only occurs but also of the interstitium. Such a testis is no longer a gland ‡ but a structure composed only of scar tissue.§ The only benefit to the patient in such a procedure over removal of the testis is the comforting feeling that there is something in the scrotal pouch.

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* For a discussion of methods of fixation after orchidopexy, reference should be made to Sievers [247] and Bruskin.[37] Attempts at establishing an artificial synorchidism [179, 274] or fixation of the testis outside the scrotum are procedures that have nothing to recommend them.

** For a discussion of results following orchidopexy, reference should be made to Bevan,[19] Dardel,[65] Goeritz,[107] Burkhard,[43] Coley,[51] Mixter,[184, 185] Caulk,[45] Paschen,[208] Brocha [33] and Tuffier.[272]

† Davison: Surg. Gynec. Obst. **12**:283, 1911.

‡ The same probably obtains when the pampiniform plexus is divided in the treatment of varicocele. Ligation and section of the veins in the suprascrotal portion of the cord not infrequently includes the internal spermatic artery.[97, 131, 132] Ligation of the veins alone, especially if done high up near the internal ring, is compatible with a testis capable of spermatogenesis, but the testis usually is reduced in size.

§ The epididymis remains fairly normal.

I would advocate, therefore, as a routine procedure in complete descent or maldescent of the testis early surgical intervention. The testis wherever possible should be brought to lie at the lowest portion of the scrotum. Only in a scrotal testis will the germinal epithelium develop normally. A high scrotal testis is better than one in the inguinal region or one retained within the abdomen. Abdominal reposition of the testis is preferable to vessel section. In such an event, the testis is probably better placed in the retroperitoneal space. If placed in the peritoneal cavity,* torsion is more likely to occur.[286] A tender or painful testis in the inguinal area that can not be gotten into the scrotum without vessel section may as well be removed, for if replaced within the abdomen not infrequently it continues to be painful.[238] An abdominal testis still maintains its internal secretion; a testis with the vessels cut is functionless. Castration in the uncomplicated undescended testis is rarely indicated. One-sixteenth of a normal testis[167] suffices to permit the secondary sex characters to develop normally in the rabbit and guinea-pig, and man probably would do very well with one. Hypertrophy of the remaining organ, however, does not occur[166] after castration. Malignancy of the testis is rare, more common in the undescended than in the scrotal testis. Scrotal fixation does not diminish the possibility of malignancy. Why remove such an organ prophylactically when it is so seldom the seat of malignancy, when we fail to apply the same rule to structures in which malignancy is not unusual?

SUMMARY

The testes of adult dogs have been placed in the inguinal region and the peritoneal cavity. Loss of the germinal epithelium always follows. These changes are not observed in the prepuberty dog's testis when placed under similar experimental conditions. Ligation of the vas deferens does not result in atrophy of the germinal cells nor in hypertrophy of the interstitium. Section of the internal spermatic artery is followed always by diminution in size of the dog's testis and by scattered degenerative changes in the tubules; spermatogenesis still continues. Ligation of the internal spermatic artery with either the anterior or posterior group of veins in the pampiniform plexus is followed by the same change that accompanies ligation of the artery alone. Ligation of the internal spermatic artery and all the veins in the pampiniform plexus always results in total destruction of the testis. The preservation of the tunica vaginalis testis is not necessary for the development of a normal gland. Replacement of the dog's testis in the scrotum after it has been resident in the peritoneal cavity or inguinal region until most of the germinal epithelium has disappeared (only spermatogonia per-

* If the testis is placed in the peritoneal cavity, the vas deferens should be divided, for should gonorrheal epididymitis occur, peritonitis would follow.

sisting) will be followed by regeneration of the mature germinal cells and normal spermatogenesis. In the adult dog only a scrotal testis is normal. The testis is very sensitive to injury and in all procedures a sufficient length of time must be allowed to intervene to preclude damage due to the manipulation alone.

Undescended testes in the human adult are practically always aspermatic. These testes, however, do elaborate the mature germinal cells, but the aberrant position of the testis does not permit of the continuance of spermatogenesis. The prepuberty testis and the testis of the new-born differ but slightly in weight and the histology is practically the same. The undescended prepuberty testis is histologically similar to the scrotal testis of the same age. If placed in the scrotum without vessel damage before puberty, the undescended testis will develop normally. Ligation of all the vessels to the testis with the exception of the artery and vein accompanying the vas deferens is followed by destruction of the testis. Scrotal fixation does not minimize the possibility of an undescended testis becoming malignant. Castration in the absence of complication is too radical a therapy in the management of undescended testes.

CONCLUSIONS

1. The undescended testis owes its imperfection to its position.
2. Scrotal fixation of the undescended testis in its physiologic position before the histologic changes incident to puberty occur will enable it to develop normally.

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